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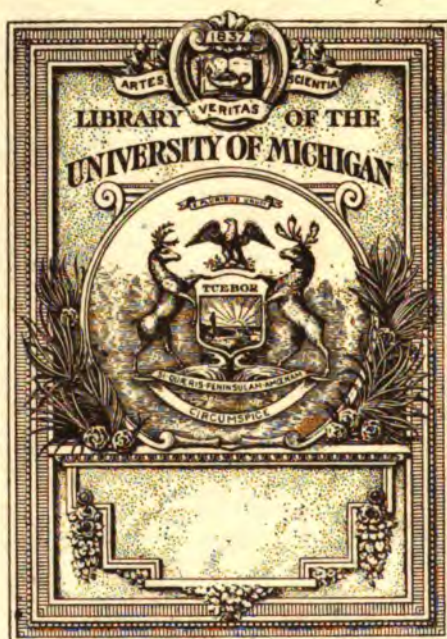
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# THE ARCHIVES OF DIAGNOSIS

A QUARTERLY JOURNAL DEVOTED TO THE STUDY  
AND THE PROGRESS OF DIAGNOSIS AND PROGNOSIS

FOUNDED AND EDITED BY  
HEINRICH STERN, MD., LL.D.  
NEW YORK



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**Special Articles**

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THE DIAGNOSIS OF DUODENAL ULCER

By JOHN B. DEAVER

Professor of Surgery, University of Pennsylvania; Surgeon in Chief  
German Hospital  
Philadelphia

The diagnosis of typical duodenal ulcer should not present any difficulty in view of the fact that the symptoms usually appear in a well-defined sequence, so well defined, indeed, that I should not hesitate to diagnose a typical case from a history given by correspondence or over the telephone, and would feel perfectly confident of having my diagnosis confirmed at operation.

The typical case history of duodenal ulcer reveals years, if not a lifetime, of attacks of epigastric discomfort after meals, that is to say, a fulness, often described as a "blown out" feeling, and a gnawing, burning sensation, rather than pain, with acid eructations, coming on from two to six, commonly three to four, hours after meals. This distress or pain, as many patients call it, rarely appears after the morning meal, but comes on with constant regularity after the heavier meals taken at noon or in the evening; the so-called hunger pain at night (about 2 A.M.) being one of the distinguishing features of the complaint. No satisfactory explanation has as yet been forthcoming as to the rationale of these hunger pains. Moynihan attributes them to changes in the muscular activity of the stomach and the duodenum stimulated by changes in the chemical quality of the chyme, especially toward the end of digestion. Food relief or subsidence of pain upon eating



or taking an alkali (soda) is another characteristic symptom. The periodicity of these attacks with intervals of complete well-being is emphasized by all authorities. Moynihan claims that they usually occur in winter and are the direct result of "cold." In my experience, and I have no doubt in that of others also, the spring and fall seasons, if any, are the ones generally mentioned.

The patients are usually middle-aged males. In an analysis of the latest series of 53 cases of duodenal ulcer operated on by me at the German Hospital of Philadelphia during the past year (January to December, 1916) there were 47 males, with an average of 41.5 years, the youngest being 21 and the oldest 63 years of age. The average of the 6 females was 36.8 years.

The physical signs consist of more or less tenderness and rigidity in the epigastric and upper right rectus regions. These, however, are of secondary importance, as it is the history mainly, and I may say exclusively, that counts in the diagnosis of the typical case.

Hemorrhage from the bowel or by mouth, as evidenced by tarry stools or by occult blood in the vomitus, is usually noted in about one-third of the cases. It was a feature in about one-seventh of the histories in this series. These patients also show a low hemoglobin percentage, though none of them presented as low a count as fifty per cent., noted by some clinicians.

Vomiting is not considered one of the commoner symptoms of ulcer of the duodenum, but it was mentioned in about one-fourth of our cases.

High acidity, that is, an excess of free hydrochloric acid, generally conceded to be pathognomonic of the disease, was noted in more than half of the cases; subacidity and normal acidity being about equally divided in the remainder.

The motility of the stomach is an important item in the symptom-complex of duodenal ulcer. That its activity is abnormally rapid is shown by the fact that in a good percentage of cases nothing of the test meal or the full meal is recovered in the usual time when the stomach is siphoned after the administration of the meal. This hypermotility of the stomach is also demonstrated by the X-ray and bismuth meal; they are thus of confirmatory rather than contributory value in the diagnosis.

Briefly stated, then, we may say that epigastric distress three or four hours after meals, relieved by eating or by alkalis; high acidity, hyperactivity of the stomach, and, in some instances, vomiting and hemorrhage are indicative of duodenal ulcer, that is, of the typical case. A correct pre-operative diagnosis was made in all but eight of the series of the present year. In one instance stone in the common duct was diagnosed in addition to duodenal ulcer and both conditions were found at operation. They were corrected by choledochostomy and posterior gastroenterostomy, the patient making an uneventful recovery. But the diagnosis is not always such smooth sailing. There is another variety—the atypical—that leads us into troubled waters. This can perhaps best be illustrated by a case taken from the series during the past year.

Male, aged 32 years, gave a history of moderate epigastric pain for one year past, coming on three or four hours after meals. The pain does not radiate; relief is obtained after eating or after vomiting. Of late the pains have increased in severity and have been coming on one-half hour after meals and have been aggravated by eating meat. There is no longer the food relief as at first, vomiting alone now affording relief. For the past six weeks the epigastric distress has been regularly accompanied by pain in the right loin near the spine, with radiations down to the right iliac fossa. Vomitus of late has occasionally been blood streaked and stools at times tarry. Urine also has sometimes of late been red. The patient complains of frontal headache and loss of weight, having lost fourteen pounds during the last two months, but seems to be gaining at the present time.

Physical examination shows a pale, sallow, anemic adult male. Abdomen: slight upper right rectus rigidity, very active peristalsis, slight tenderness on deep palpation, especially in the right loin near the spinal column and over McBurney's point.

At operation a duodenal ulcer was found welded together with the great omentum, the hepatic flexure and the pancreas. The appendix was bound down with its tip in the subcecal fossa. The appendix was removed; a posterior gastroenterostomy was done; the duodenum was not plicated.

The patient left the hospital in excellent condition without any evidence of gastric disturbance.

These atypical cases more often simulate appendicitis, especially where the appendix is high, than other conditions from which they can with more or less ease be differentiated, such as gastric ulcer, cholelithiasis, cholecystitis, chronic pancreatitis and pancreatic lymphangitis.

Chronic appendicitis frequently presents the same hunger pains as in duodenal ulcer, hyperacidity is not unusual and many cases show the same chronicity as in duodenal ulcer. The main difference between the two is the freedom from discomfort in the duodenal ulcers between the attacks, while in appendicitis the flatulency and discomfort are apt to be constantly present. But these patients with "appendiceal indigestion" usually suffer more pain after certain kinds of food, especially starchy food and red meats. The pain, however, usually is not so severe as in duodenal ulcer and radiates downward. The latter being one of the main points in the differential diagnosis. In appendicitis exercise frequently increases the local discomfort—not so in duodenal ulcer. In fact, the appendix is found diseased in so many cases of duodenal as well as of gastric ulcer, that these latter may be considered secondary conditions; that is to say, the result of infection from some other organ with the evidence strongly in favor of the appendix as the *corpus delicti*. I, therefore, make it a practice to remove the appendix in practically all cases of gastric and duodenal ulcer.

Some authors claim that it is almost impossible clinically to differentiate between gastric and duodenal ulcer, but it seems to me that there are enough points of variation to enable such a differential diagnosis with some degree of certainty. In distinguishing between the two we may to some extent be guided by the time relation of the ingestion of food and the onset of the symptoms. Although the chain of symptoms of duodenal ulcer is said to be not much affected by the location of the ulcer, it is generally conceded that the longer the interval between the meals taken and the appearance of the pain and the more prompt the food relief, the lower down will the ulcer eventually be found. Therefore, if pain appears soon after eating, in one-half to two hours, and the food relief is not prompt, we may logically expect to find a gastric rather than a duodenal location. Again the radiations of pain, if any, in duodenal ulcer are usually to the right, while in gastric ulcer the pain

radiates to the left as a rule. The pain also is apt to be more constant than in duodenal ulcer. Einhorn's duodenal bucket has been found useful in some instances in approximating the location of the ulcer with more or less precision, but I have not introduced it as a routine procedure in my service at the German Hospital of Philadelphia. Vomiting is also more frequently a symptom of gastric ulcer, as is also hemorrhage, the latter usually in the form of hematemesis, while in duodenal ulcer it is more generally melenic.

Cholelithiasis presents rather more difficulty, but care in taking the history will usually enable the experienced clinician to forecast the true state of affairs. The diagnosis is oftentimes uncertain when adhesions exist between the gall-bladder and the stomach and the duodenum, or when the gall-stones have pushed toward the duodenum; hyperacidity being also a symptom of gall-stone disease, adds to the confusion. On the whole, however, cholelithiasis is marked by such severe colicky pain with sudden and unaccountable onset, and almost as sudden and mysterious cessation, that recognition should, as a rule, be easy. Lavage will frequently cut short an attack of biliary colic, but has no influence on the pain of duodenal ulcer. In this connection Moynihan mentions the gastric crises of tabes dorsalis, as a possible source of error in diagnosis.

Chronic cholecystitis very frequently clouds the diagnosis of ulcer, especially of the perforating duodenal ulcer. It presents the same chronicity, though the attacks do not last so long, the pain, hyperacidity and flatulency also present show a certain degree of relationship to food intake, while not infrequently the absence of typical jaundice in cholecystitis and its presence in duodenal ulcer, as noted in several of our cases, make confusion worse confounded.

Symptoms similar to those of chronic pancreatitis or some pancreatic involvement, such as pancreatic lymphangitis, are not rarely met with in duodenal ulcer. This is not surprising in view of the close relationship existing between the duodenum and the pancreas and the frequent infiltration of ulcer into the pancreas itself and the close intercommunication between the pancreatic and the duodenal lymphatics. For example, loss of weight and strength, a fairly constant clinical feature of chronic pancreatitis, was recorded in one-third of our cases during the past year. The character of the pain in chronic pancreatitis is moderate, as it is in the ma-

jority of duodenal ulcer cases, and there is the same epigastric oppression. A valuable distinguishing feature, however, is the absence in the pancreatic disease of any definite relation to eating or drinking, or to the kind of food taken.

Malignant neoplasms of the intestines, in their early stages, sometimes simulate the symptoms of duodenal ulcer, but careful inquiry will usually elicit the fact that the attacks, though presenting the same periodicity as in duodenal ulcer, bear no relation to food, neither in their onset nor in the relief of symptoms. In these atypical cases, however, nothing short of incision and inspection will enable us definitely to determine the nature of the lesion.

Two cases of the present series diagnosed as acute appendicitis both proved to be subacute perforating ulcers which had been closed by plastic exudate. A pre-operative diagnosis of chronic appendicitis in one instance proved correct, but an ulcer of the duodenum was also found.

In four cases a clinical diagnosis of gall-bladder disease was made and in two ulcer of the duodenum was present in addition to the cholecystitis. In one case the diagnosis wavered between carcinoma and ulcer of the duodenum; operation revealed the latter.

In thus giving a cursory summary of the main diagnostic points of ulcer of the duodenum, I may say, I hope, that I speak from a wide experience. During the past six years I have treated by operation four hundred cases of diseases of the stomach and duodenum, of which two hundred were duodenal ulcers. With your permission, I should like to say a few words with regard to the treatment of these ulcers. I know that I risk incurring the displeasure of the internist when I say that he is responsible for a large number of cases of malignant disease of the gastrointestinal tract by attempting medical treatment of these gastric and duodenal conditions for any prolonged period of time. It would take me too far afield to enlarge upon the likelihood of malignant degeneration of these ulcers, especially those of the stomach. Suffice it to say that the percentage is variously estimated to be from thirty-five (my cases) to seventy per cent. (other authors). Add to this the fact that in our latest series of cases three specimens of duodenal ulcer were returned from the pathological laboratory bearing the legend "incipient malignancy," and you will realize why I make

this statement. A serious and more frequent menace presented by duodenal ulcers is perforation and hemorrhage. I find from my statistics that perforation takes place in about fifteen per cent. of the cases and that fully eighty per cent. give a history of previous gastric disturbance. I have had only one death from hemorrhage in these cases. This patient refused operation at the opportune time and died of hemorrhage from the bowel while still in the hospital. At autopsy an ulcer was found on the pancreatic side of the second portion of the duodenum.

With an operative mortality of 3.7 per cent. in the chronic duodenal ulcers and only one death among forty-six perforated cases (thirty-six recent statistics at the German Hospital and ten cases of an earlier series), surely it is, to say the least, unjust to subject these patients to the discomfort and the risk of recurrence and the more serious dangers already alluded to.

We were able to trace about thirty per cent. of the cases of perforated duodenal ulcer cases, all of whom reported well without return of symptoms; the others reported occasionally epigastric distress after eating. One case gave a history of hemorrhage from the stomach due to exertion, this took place three years after operation.

As to the type of operation. Incision and drainage of sub-diaphragmatic abscess was found sufficient in one of the perforated ulcer cases, in all the others a posterior gastroenterostomy was performed. In thirteen instances this was the only procedure; in seventeen it was combined with pylorotomy, and in one case in which there was also a gastric ulcer located on the lesser curvature of the stomach necessitating a subtotal gastrectomy. In ten cases the ulcer was invaginated and in five it was excised; plication of the duodenum was done twice. In accordance with our usual procedure, the appendix was removed in all cases, except where it had already been removed at a previous operation. In one other case, besides plication of the duodenum, the ulcer was invaginated. In the cases complicated with cholecystitis a cholecystectomy was performed in two instances, combined with a choledochostomy in one case. A choledochostomy, already mentioned, was done in one case with stone in the common duct.



THE POSSIBLE DEPENDENCE OF GASTRIC AND DUODENAL ULCER IN MAN ON A DISTURBANCE OF INTERNAL SECRETIONS

By G. A. FRIEDMAN

Adjunct Professor of Clinical Medicine, New York Polyclinic Medical School and Hospital; Gastroenterologist, Gouverneur Hospital  
New York

The explanation of a disease is plausible when the results of experimentation and clinical experience are in accord with the findings of the pathologist. However, since the pathologist has rarely the opportunities to investigate the changes that occur during the initial stage of an illness when the pathological lesions are first being developed, and since he deals in the majority of instances with alterations of tissues probably existing an entire life time and consequently when the disease is most advanced and the pathological changes so vastly modified by numerous factors, the suggestions as to the possible explanation of the pathogenesis of certain diseases may come from the experimenter and the clinician. This is apparently true in regard to the origin of gastric and duodenal ulcer, for lately it has been shown that the experimenter and the clinician have been the real investigators of this disease, and have rendered possible an explanation of the common cause of peptic ulcer in man by a study of internal secretions, based on experimental work which seems to harmonize with clinical experience.

It is beyond my province to discuss the theories that have been advanced to explain the pathogenesis of ulcer in man, but suffice it to say that not one of the explanations has been accepted as the common cause of ulcer, and this may be seen from a statement in a circular of the Special Committee in Germany for the collective investigation of gastric ulcer. "The nature of gastric ulcer," the statement reads, "has not yet been explained, even if some have been successful in producing it experimentally with all its characteristic signs. The etiology of ulcer in man is practically unknown." However, by our increased knowledge of activity of endocrine glands, by our closer study of the anatomy and the physiology of the nervous system, by the careful investigation and observation of cases by the clinician who has had the opportunity of watching and

studying individuals during the whole course of the disease, both medical and surgical, and by our improved methods of animal experimentation, knowledge of real value has been obtained. An attempt is, therefore, made to explain the pathogenesis of ulcer in man from a different standpoint.

Briefly mentioned, the theories that have been advanced in previous years, are the vascular, the neurogenous, the toxic and the microbic. Perhaps a few words should be said concerning the neurogenous origin of ulcer. It is a well known fact that ulcers or erosions of the stomach were obtained by cutting the vagus nerve, the sympathetic, or both. In Lichtenbelt's<sup>1</sup> experiments, the ulcers produced by vagotomy persisted without tendency to heal, and on account of the absence of this tendency, they greatly simulated the peptic ulcer. Still the neurogenous explanation of peptic ulcer never obtained due prominence, probably because the clinician did not appreciate the nervous symptoms found in individuals afflicted with ulcer, and because the pathologist was rarely able to discover changes in the nerves of his ulcer material. But we now know that a functional disturbance in one structure may produce pathological changes in another; and in seeking the origin of the initial lesion in peptic ulcer, we have met with many strong proofs, both clinical and experimental, which have led us to conclude that the lesion is due to a functional disturbance of the nerves supplying the parts involved. This functional disturbance of the nerves may probably arise from errors in internal secretion.

Due to an irritation arising from some source in the body, probably the disturbed secretions of the ductless glands, or produced by the injections of certain substances or drugs into the body, stimulation of one of the nerves leading to one or more of the smaller gastric or duodenal arteries, may develop a spasm of that vessel or cause occlusion of the vessel by a spastic contraction of the musculature surrounding it. The result from either of these possibilities is an ischemia of the gastric mucosa supplied by the vessel involved and thus, we have the initial lesion. Since cardiospasm, gastrospasm, pylorospasm, and hour-glass stomach are known to exist in individuals without the presence of an organic lesion as an ulcer, a primary irritative state of the vagus nerve, with the resulting gastric muscular spasm, cannot be denied. If such irritative conditions

arising from functional disturbances in nerves are true for large areas of musculature, then it is probably also true for minute areas.

The explanation here set forth—that of spastic ischemia—must not be confused with the vascular theory by which an attempt is made to explain the origin of ulcer as arising from a stasis in the gastric vessels with the resulting changes in nutrition, or affliction of the blood vessels by disease, by embolic or thrombotic processes or by arteriosclerosis. It is true that these occasionally cause ulceration, but they are by no means the common causes. The theory that ulcer is caused by an irritation of the nerve leading to the smallest vessel causing a spasm of its wall, or even its occlusion by spastic contraction of the musculature surrounding it, either condition leading to an ischemia of the mucosa with subsequent ulceration, is favored by such men as Lebert,<sup>2</sup> and recently by v. Bergman,<sup>3</sup> Benecke<sup>4</sup> and others.

The organs which are supplied with smooth musculature as the stomach or duodenum, are under control of the vegetative nervous system, the regulation of which is partly independent of the central nervous system. Langley divided this vegetative system into the cranial-sacral-autonomous group, extended vagus, or the para-sympathetic group and the sympathicus proper. He also showed that the antagonistic physiological relation of both groups existed not only in the heart, where this antagonism was most evident, but was present in the stomach and intestines where, however, the vagus fibers caused stimulation of the smooth musculature and the sympathicus inhibition of peristalsis and secretion.

Langley's well-known teachings of antagonism have been strengthened by the researches of H. H. Meyer<sup>5</sup> who showed that certain drugs as pilocarpin, muscarin, physostigmin, and cholin have a selective action upon the autonomous nervous system and also upon the sweat glands. Atropin paralyzes this system. Adrenalin, however, has been shown to have a stimulating effect on the sympathetic nerves. There is as yet no known paralyzer of the sympathetic similar to the action of atropin on the extended vagus or autonomous system.

The vegetative nerve system supplies the glands of internal secretion as well as the viscera. The stomach is supplied by the terminal branches of the vagus. The duodenum is largely supplied by the

sympathetic fibers and by some of the vagus fibers. The adrenals obtain their nerve supply almost entirely from the sympathetic through the splanchnic nerve. The thyroid, however, has a double innervation, the sympathetic and the vagus. The pituitary body may be excluded from consideration here since the secretion of this gland acts upon tissues innervated by the pelvic nerve.

There now remains the application of these physiological, pharmacological, and anatomical facts to the clinic and this was done by Eppinger<sup>6</sup> and Hess,<sup>7</sup> whose teachings of vagotonia and sympathicotonia are well known. They have clinically divided patients into two groups, the vagotoniac and the sympathicotoniac, basing their classification upon the different symptoms arising from disturbances in one or the other of the subdivisions of the vegetative system. By a vagotoniac is meant an individual who is extraordinarily susceptible to drugs, stimulating or paralyzing the vagus nerve, as pilocarpin, atropin. Sympathicotoniacs on the other hand, are extremely susceptible to adrenalin, the stimulating drug par excellence of the sympathetic. The symptoms and signs belonging to each clinical group and the symptoms produced by the injection of the specific drugs mentioned, have been well described and need not be mentioned here. However, it must be remembered that no sharp lines can be drawn between the two groups, for often the symptoms overlap, but one should always consider the symptoms and signs related to vagotonia and sympathicotonia, as stigmata of the vegetative nervous system.

In order to make use of all the considerations mentioned, it must be shown that vegetative stigmata are actually present in the majority of individuals suffering from gastric and duodenal ulcer. Clinical experience shows that patients suffering from chronic peptic ulcer may be divided into two groups:—First, those in whom the organic element without apparent nervous manifestations is evident. At operations, ulcer is usually found. These patients are frequently benefited by the various surgical procedures. Secondly, those in whom the nervous element is so predominant that it is often difficult to eliminate the organic element as the chief cause of trouble. At operation, ulcer is found, but usually the result of the operation is not beneficial. If one questions these patients carefully as to symptoms and signs regarding the nervous system, autonomic

and sympathetic, he will often be surprised as to the number of vegetative stigmata present, even in the majority of cases belonging to the first group. The complete absence of symptoms and signs pointing to a disturbed equilibrium of the vegetative nervous system may be mainly noticed in the fifth or sixth decade of life. The younger the individual afflicted with peptic ulcer, the more numerous are the vegetative stigmata. But even in old people, in whom the first symptoms of ulcer dates back twenty or twenty-five years, one will elicit the history of previous nervous symptoms which may be interpreted as evidence of vegetative disturbance. It is a well-known fact that functional disturbances have a tendency to disappear with age, though the organic lesion, the sequel of such disturbances may remain.

The symptoms and signs usually found in cases of peptic ulcer are practically similar to those found in vagotonia and sympathicotonia, the underlying causative factor being the same, namely a disturbance in the vegetative nervous system. Gastric ulcer cases show frequently symptoms of vagotonia, duodenal ulcer cases more often symptoms of sympathicotonia. The chief symptoms elicited in the history are:—excessive salivation or dryness of the mouth, clammy or dry hands and feet, a tendency to perspiration, usually localized to some area, as the axilla, beneath the breasts, etc., constipation usually of the spastic type or rarely diarrhea. Among the more important physical signs and phenomena in ulcer cases suggestive of a derangement of the vegetative system we find narrow or wide pupils, exophthalmos or various grades of protrusion of the bulbi, even exophthalmos, absence or diminished corneal reflex, narrow or widened palpebral fissures, excessive flow of tears and glittering eyes, gastric succorhea or achylia, high or low gastric acidity, gastrointestinal hyper or hypomotility, spastic or atonic constipation, bradycardia or tachycardia, absence or exaggeration of gag reflex, dermatographia and Ashmer's oculo-cardiac reflex, which is produced by a continuous pressure with the fingers on the eyeball and noting the sudden slowing of the pulse rate, a sign of vagotonia. In addition, we can note in cases of peptic ulcer the presence of Stiller's habitus and as pointed out by E. Kraus,<sup>8</sup> the so-called Blähhals, a prominence of the neck due to an extreme vascularization of the thyroid gland.

Furthermore, the patients with peptic ulcer respond to the pilocarpin and adrenalin tests. Westphal<sup>9</sup> and Katsch,<sup>10</sup> who have made an extensive study of this subject, pointed out that some of these patients are more susceptible to pilocarpin and others to adrenalin, which fact proves the clinical findings of Eppinger and Hess. Only in the middle-aged patients they found frequently that the reaction was negative to either of these drugs. My own experience has taught me that individuals suffering from gastric ulcer present more often vagotonic symptoms and respond rather strongly to the injections of pilocarpin, while those suffering from duodenal ulcer show frequently the sympathicotonic symptoms and react therefore more often positively to injections of adrenalin. These facts are probably explained by the differences in innervation of the stomach and duodenum. We see, therefore, that clinically and pharmacologically the symptoms and signs which define the status of the vagotonic and the sympathicotonic may be found in patients suffering from gastric and duodenal ulcer.

There are, moreover, other factors which suggest the dependence of ulcer on the derangement of the vegetative system with the consequent disturbance in certain ductless glands. Several years ago, Dr. Lewis, of the General Memorial Hospital, assistant of Professor Benedict, at my request, has examined the percentage of blood sugar in a number of my patients whom I believed had peptic ulcers. Operative evidence was obtained in ten of them, and in five of these, callous duodenal ulcer was found. In these the percentage of blood sugar was above normal, while in four others who had pyloric ulcers, the percentage of blood sugar was below normal. However, in the tenth, in whom ulcer of the lesser curvature was found, the percentage of blood sugar corresponded to that found in duodenal ulcer cases.

The change in the percentage of blood sugar is, I believe, due to a glandular disturbance. That the product of the secretion of the islands of Langerhaus requires the presence of some other agent for its glycolytic action was first shown by Diamare,<sup>11</sup> and later by others. Sajous,<sup>12</sup> in 1907, suggested that the secretion of the adrenal was the necessary factor in this process. This was further substantiated by W. G. MacCallum<sup>13</sup> four years later.

Secondly, we have the characteristic differences in the blood pic-



tures between chronic callous gastric and duodenal ulcers. These differences have been noted and pointed out by me in several of my published papers.<sup>14, 15, 16, 17</sup> But the significant fact is the resemblance of the blood count in duodenal ulcer to that of polyglobulia induced by adrenalin injections and of the blood picture in gastric ulcer to that found in hyperthyroidism. Bertelli,<sup>18</sup> Falta,<sup>19</sup> and Schweeger<sup>20</sup> and later Imachnitzky<sup>21</sup> have observed an increase of erythrocytes of from 30 to 100 per cent. in dogs and man after intravenous or subcutaneous injections of epinephrin (this experimental polyglobulia lasting sometimes for about 30 hours), and they have also noted after such injections a marked decrease in the eosinophiles. In my papers I show a polyglobulia and an eosinopenia to be significant of duodenal ulcer of the callous type. The similarity of the blood picture of pyloric ulcer to that of hyperthyroidism—a mononucleosis and a relative eosinophilia—is also quite marked. Kocher<sup>22</sup> has repeatedly stated that the mononuclear cells frequently predominate in Graves' disease. In addition, Eppinger<sup>23</sup> frequently found an increase of eosinophiles. I have observed in nearly every case of the callous pyloric ulcer an increased number of small mononuclears and an increase in the eosinophiles. Although some state that in myxedema there is an increase in mononuclears, others believe that the picture found in this condition is not analogous to that in Graves' disease, because after the administration of thyroïdin in myxedema, the blood picture returns to normal, while in Graves' disease it diverges still further from normal. It should be mentioned here that Kaufmann<sup>24</sup> who made blood examinations in a number of gastrointestinal disorders has found frequently lymphocytoses.

Although we have recently learned to associate sympathicotonic and vagotonic symptoms with disturbances in equilibrium of the vegetative nervous system and although we have seen that the occurrence of ulcer is frequent in cases of vagotonia and sympathicotonia, there remains the correlation of these facts with disturbances in internal secretions. It is well-known that the vegetative nervous system is the regulator of the glands of internal secretions as well as the visceral organs. Asher<sup>25</sup> and Flachs<sup>26</sup> have indisputably shown that the thyroid gland is influenced by the superior and inferior laryngeal nerves. Biedl,<sup>27</sup> Dryer,<sup>28</sup> Tsherboksaroff<sup>29</sup> and

Asher<sup>30</sup> have in addition shown with absolute certainty that the splanchnic nerve is the secretory nerve to the adrenal. It appears that when one of the glands, for instance, the thyroid or adrenal, has been stimulated by the vegetative nerves and kept under its influence abnormally, there may be a reaction, and the nerve itself may become influenced through the disturbed activity of the gland; in other words, a vicious circle may set in between the vegetative nerve and the gland. Since, therefore, a disturbance in the vegetative nerve, vagus or sympathetic, may lead to disturbances in the function of the thyroid or adrenal, and since there is a reciprocal reaction between them, a minor degree of disturbance in these glands may show its influence upon the nerves, and the result of such influence may become evident clinically, by pathological changes in the structures supplied by the nerves affected, and by the production of the symptoms and signs of vagotonia or sympathicotonia. The mode by which the ductless glandular system may influence the excitability of the vegetative nerves is not quite known, but it is probably produced through the agency of hormones. At least this has been established with certainty in regard to adrenalin, and it is possibly true of the other internal secretions.

The vegetative stigmata found in individuals suffering from peptic ulcer, the susceptibility of these patients to the administration of drugs which have a selective action upon the vagus and sympathicus, the blood picture of duodenal ulcer resembling the experimental polyglobulia and eosinopenia after adrenalin injections, the blood picture in pyloric ulcer which frequently simulates that found in hyperthyroidism, all these facts are hardly sufficient to lead us to a conclusion as to the possible dependence of ulcer in man on a disturbance of secretion of certain ductless glands. Thus far we know that an excess of adrenalin is circulating in the blood of the sympathicotonic. The excess of secretion which is supposed to circulate in the blood of the vagotonic is not known. Eppinger and Hess have named this hypothetical secretion "automin" and this is supposed to stimulate the autonomous system. However, since the thyroid has two secreting components, one from the sympathetic and the other from the vagus, the component originating through stimulation of the vagus fibers may be the one circulating in the blood of the vagotonic. The experiments of Westphal<sup>31</sup>

seem to hint to such a possibility. He first tried to imitate vagotonia in rabbits, cats, dogs and guinea pigs by injections of pilocarpin. He succeeded in producing peptic ulcer of the stomach in nearly all of the rabbits, and in some of the cats and in dogs. I have repeated his experiments on rabbits and present here four specimens showing distinctly peptic erosions after pilocarpin injections. Since, after thyroidectomy, as it has been proven, pilocarpin had little effect in provoking vagotonic symptoms, we possibly have proof of the relation rather indirectly of some component of the thyroid secretion upon the vagus. This leads me to mention the so-called vagotonic type of Graves' disease. This has first been described by Eppinger and Hess who have shown that, although it is generally acknowledged that Graves' disease is due to a disturbance in the sympathetic, there are definite forms of Graves' disease with symptoms and signs pointing to a heightened tone of the vagus element supplying that gland.

We became more convinced that an excess of thyroid secretion may affect the mucous membrane of the stomach, producing erosions, since in two dogs and one rabbit (of four animals experimented upon by subcutaneous and intravenous injections for about one week of dessicated thyroid gland) we have obtained gastric erosions.

✓/ Seeing a possible connecting link between the polyglobulias and eosinopenia which I found in duodenal ulcer and the experimental polyglobulias found after injections of adrenalin on the one hand, and the tendency of adrenalin to affect tissues innervated by the sympathetic on the other, we set up the working hypothesis that the initial lesion of duodenal ulcer may be caused by excessive secretion of the adrenals. With this object in view experiments were undertaken consisting of repeated injections of adrenalin in dogs. Since some of the experiments described here have been fully published in two of my papers,<sup>32, 33</sup> I need but mention them briefly.

It was found that injections of adrenalin administered to dogs intravenously, subcutaneously, or intramuscularly for about one to two weeks in dosages not exceeding three milligrams of the usual commercial solution (1:1000) are liable to cause lesions, erosions or superficial ulcerations in the duodenum. As such lesions were found in the duodenal mucosa in eleven dogs out of twelve and

later in two out of four and only occasionally gross changes were noted in other organs, we concluded that adrenalin might have a preferential action upon the duodenum, probably because of the latter's rich sympathetic nerve supply, since as has been pointed out, the sympathetic. resp. splanchnicus is the secretory nerve of the adrenals. In normal dogs, autopsied as controls, the gastric and duodenal mucosa was found to be intact. In looking over the experiments with injection of adrenalin, in rabbits which were done previously for other purposes, we were surprised to find frequently notes of "marked congestion" or lesions in the first portion of the duodenum.

After one-sided thyroidectomy in dogs and in rabbits, lesions or ulcers were found in the duodenum more frequently than in the stomach. Occasionally ulcers were found in both viscera or in the jejunum. A careful search in the literature has revealed that Carlson<sup>34</sup> and Jacobsohn<sup>35</sup> have incidentally found gastric and intestinal lesions in seventy-five per cent. of thyroidectomized dogs. They emphasize the fact that the ulcers were always most extensive in the upper part of the duodenum.

After extirpation of both adrenals in two stage operations and after extirpation of one adrenal in dogs and in rabbits and also later in cats and in guinea pigs, lesions or erosions were frequently found in the stomach. In the duodenum of rabbits and guinea pigs and in one dog, lesions were found also after one-sided adrenalectomy, when the unextirpated adrenal became hypertrophied.

Gastric ulcers after extirpation of adrenals were first produced by Finzi.<sup>36</sup> He also showed that if after extirpation of the suprarenal gland, adrenalin is injected, the gastric mucosa remains intact. Elliot<sup>37</sup> produced upon adrenalectomy, ulcers in cats, and recently Mann<sup>38, 39</sup> in dogs and in cats.

After extirpation of one adrenal and one thyroid lobe of the same or the opposite sides, no lesions in the stomach or duodenum were found in our experiments.

To sum up the results of my experiments presented in the two communications and from those yet unpublished, we have:  
1. Adrenal insufficiency causes in various species of animals, lesions or ulcers in the stomach. 2. An excess of thyroid gland, as produced by repeated intravenous injections, was probably responsible

for the gastric lesions in three animals out of the four experimented upon. 3. Thyroid hypofunction caused the appearance of duodenal and gastric lesions. 4. An excess of adrenalin produced by repeated injections of the drug, led to appearance of lesions in the duodenum of dogs and rabbits. 5. The simultaneous production of adrenal and thyroid hypofunction did not lead to any lesion in the stomach, nor in the duodenum of rabbits. 6. When after removal of an adrenal the other became occasionally hypertrophied, lesions were seen in both viscera in rabbits.


From our experiments it seems probable that gastric lesions may be dependent upon adrenal insufficiency as well as upon excess of adrenalin. Gastric and duodenal lesions may be dependent upon the alternating effect of hypo- and hyper-function of the adrenals. From all these considerations a correlation of secretions of the thyroid and adrenals seems to be plausible in the causation of gastric and duodenal lesions in our animals.

For the sake of briefness, I have to omit a discussion, presented in one of my papers as to reciprocal relations between the thyroid and the adrenals as obtained in our animals. In our thirty-six thyroidectomies performed in dogs and in rabbits, we have never observed hypertrophy of the unremoved thyroid lobe unless as happened in several dogs in which infection set in. But we did observe in two dogs and two rabbits after parathyroidectomy where parathyroids were not spared, a marked hypertrophy of the adrenals. These animals died from tetany. It is possible then, that after one-sided thyroidectomy, the adrenals hyperfunctionate without hypertrophy and in consequence duodenal lesions frequently develop as they do after injections of adrenalin. The gastric lesions after removal of the adrenals may be due to a hyperfunction of thyroid as occurs after injection of dessicated thyroid extract. Although, as has been mentioned, there is a vagotonic type of Graves' disease, Higier<sup>40</sup> believes that Addison's disease also presents the best illustrative instance of a slowly developing vagotonia, emaciation, diarrhea, low blood pressure, and the reduction or disappearance of blood sugar. In classifying Addison's disease, therefore, as vagotonia, Higier sees the abolishment of the most important sympathetic impulses. If a vagotonic type of Addison's disease is possible, what should be our conception of it? As Addison's

disease develops, the sympathetic impulses become gradually abolished, since the normal tone of the sympathetic is under control of the adrenals. With the double innervation in the thyroid, the antagonistic vagus element in the gland, therefore, hyperfunctionates, and as a result an excessive amount of thyroid products, the so-called autonomins of Eppinger and Hess, will be discharged into the circulation. The effect, however, of this disturbance in secretion, may become modified by the presence of diseased adrenals. Just as there is a possible connecting link, therefore, between vagotonic exophthalmic goiter and Addison's disease, a somewhat similar correlation might have developed in our animals after removal of one adrenal and one of the thyroid lobes.

We now turn to the applicability of these experiments to the clinic. Since vegetative stigmata are found in the majority of patients suffering from gastric or duodenal ulcer, and since a disturbance of the equilibrium in the vegetative nervous system may lead to a disturbance in the secretion of the thyroid or adrenals or both, there is a possible connecting link between the vegetative stigmata and the appearance of the initial lesion of peptic ulcer. Although Eppinger and Hess were the first to recognize anomalies of constitution as dependent on a derangement of the vegetative nerves, it was Körte<sup>41</sup> who originally in a discussion at the XXXV Congress of the German Society für Chirurgie in 1906 correlated the anomalies of constitution with peptic ulcer. He then expressed his opinion that a local affection of the gastric mucous membrane might possibly bear some relation to the constitutional anomalies, the nature of which was unknown, and that these anomalies of constitution were the disturbing factors in the healing process of peptic ulcer.

However, the functional disturbance in the vegetative system must not necessarily lead to a pathological change in the ductless glands as in the vegetative organs. As often happens, an organ neurosis, gastric or intestinal, without organic phenomena may be the result. Hence we may also assume a ductless gland neurosis by which I mean a functional derangement of the nerve supply to the gland without a pathological change in that organ or nerve but leading, however, to a disturbance in secretion. Bauer,<sup>42</sup> Hemmeter<sup>43</sup> and others dealt extensively with these neuroses of endocrinous glands.



The secretion of such a disturbed gland may react upon the vegetative nerve system and influence the system still more, thus establishing a vicious circle as has been explained previously. These factors may lead to the anomalies in constitution which Eppinger and Hess and Körte have noted, and may also produce the initial lesion of the ulcer. With the return to normal conditions in the vegetative nervous system and in the glands, the anomalies in constitution may disappear but the ulcer, however, the material or pathological result of all these disturbances, remains, and may now proceed to heal. McCallum<sup>44</sup> points out that many ulcers in man heal spontaneously as may be judged from the scars at autopsies. All have seen at operations deep scars in the stomach or duodenum from healed ulcers. Ulcers heal, therefore, when the anomalies of constitution due to disturbance in the equilibrium of the vegetative nerve system, and hence in the disturbance of the glands, disappear, but the scar is the witness, however, that such a disturbance has existed. One may now comprehend why some patients do not show vegetative stigmata at the time of examination, but have all the evidence pointing to an organic lesion, and why such patients are fully benefited by surgical procedures.

✓ If it be true that the initial lesion of peptic ulcer is due to anomalies of constitution then the occurrence of peptic ulcers in the newborn and in young, may be explained as congenital—that is to say the inheritance of the anomalous condition. Huber<sup>45</sup> has made a special study of the occurrence of peptic ulcer in several members of many families. He came to the conclusion that the occurrence is not rare. I have under observation a girl with gastric ulcer whose mother was operated for gastric ulcer one year ago. I am almost certain that the more we question our patients in regard to inheritance, the more frequently will we discover the presence of the ulcer running in the same family.

There is no doubt that chronicity of ulcer depends upon many factors. The healing process of ulcer is probably prevented in man by anomalies in constitution which is difficult to install in animals. The spastic ischemia results in the initial lesion. Through the corrosive action of the excess of hydrochloric acid the further development of ulcer occurs. The acidity plays undoubtedly a rôle as a secondary factor. The fact that in peptic ulcer one may find normal

acidity hypo-acidity or even achylia gastrica does not prove anything to the contrary. We know that the vagotonic shows in his stomach hyperchlorhydria, but according to Eppinger and Hess the vagotonic might become a sympathicotonic in whom low acidity is usually found as a result of the change. Aschoff's<sup>46</sup> explanation that the chronic character of ulcer, its location, and its shape, depend primarily on the mechanical conditions and the prolonged contact with gastric juice at the physiologic points of narrowing of the stomach and also upon the mechanical friction and stress at the lesser curvature along which the ingesta travels to the pylorus, is probably true. He emphasizes that the chronicity of ulcer does not depend on primary disease of the blood vessels.

The question now naturally arises, since the acute gastric and duodenal ulcer, the initial lesions from which the chronic ulcers may develop, are produced experimentally through a disturbance of thyroid and adrenal, why are ulcers not found in Addison's disease, in myxedema or in exophthalmic goiter? As to Addison's disease one must say that gastric disturbances do occur frequently. There is a special gastric type in this disease. Ulcers probably do not develop because Addison's disease is in the majority of instances a tuberculous condition. That tuberculosis has little affinity for the stomach and the first portion of the duodenum may be surmised by the fact that tuberculous ulcers of the stomach or duodenum are extremely rare in spite of the fact that tuberculosis is a common disease. Moreover, in individuals with gastric or duodenal ulcer, the adrenals may be found affected, as seen from the pathological findings of Finzi,<sup>47</sup> who showed the adrenals to be markedly affected in five necropsies. I have seen several cases of Graves' disease in women in whom symptoms of peptic ulcer were present. I have also recently observed in two female patients who had been operated for exophthalmic goiter, developing later clear symptoms and signs of peptic ulcer.

✓ The increased frequency of peptic ulcer is a well-known fact and this is certainly due to the mode of living—the hurried life, the quick lunches, the tremendous business worries, and generally the wear and tear of life. That this nervous tension may lead to disturbances in equilibrium of the vegetative system is beyond doubt. The relation of this disturbance to the hyper-, hypo- and dys-func-



tion of the thyroid and adrenals, and reaction of the secretions of these glands in turn upon the vegetative system with production of constitutional anomalies, have been explained. The presence of ulcer in these cases, the production of ulcer experimentally under conditions similar to that found in man, point to the initial lesion of the condition as due to disturbances in internal secretions.

Although it cannot be denied that the causes of the initial lesions of ulcer are manifold, the explanation set forth, however, appears to be by far, the most frequent cause of ulcer.

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## THE PROGNOSIS OF DUODENAL ULCER

BY MAX EINHORN

Professor of Medicine at the New York Postgraduate Medical School  
New York

The prognosis of a disease runs parallel with its therapeutic possibilities. Progress in the cure of a malady improves its prognosis. In duodenal ulcer great advances have recently been made in diagnosis as well as in treatment. The outlook, therefore, for patients suffering from a duodenal ulcer is nowadays much brighter and more favorable than in former years.

With regard to prognosis it will be well to divide duodenal ulcers into the following groups:

- (a) Simple duodenal ulcer;
- (b) Duodenal ulcer accompanied by pylorospasm and usually also hypersecretion (alimentary or continuous);
- (c) Duodenal ulcer accompanied by pyloric or duodenal stenosis;
- (d) Duodenal ulcer with recurrent hemorrhages.

(a) Simple duodenal ulcer. Here the usual symptoms are epigastric distress two to three hours after meals; sometimes "hunger pain"; long periods of euphoria alternating with comparatively short periods of suffering. Gastric hemorrhage or melena may have occurred once.

This group gives a comparatively good prognosis provided that some form of a rest cure is rigidly carried out: rectal alimentation then von Leube-Ziemssen milk diet; or duodenal alimentation; or simply a milk and egg diet and rest abed for about two to three weeks. Later on no over-exertion (physical or mental) and a general hygienic way of living.

The oftener the attacks recur the more doubtful the prognosis becomes as to a complete cure by medical measures. Operative intervention (gastroenterostomy preferably with pyloric occlusion) offers a pretty good prognosis with regard to the future.

(b) Duodenal ulcer accompanied by pylorospasm and also hypersecretion (alimentary or continuous). Severe pains and frequent vomiting are here the chief symptoms. Hypersecretion either alimentary or continuous is here constantly encountered. The gastric juice is usually hyperacid. When the pylorospasm reaches a higher

degree slight isochymia appears off and on. The prognosis of this group is quite severe under ordinary methods of treatment (alkalies, even milk diet). Duodenal alimentation gives a better prognosis. But in case the latter does not produce the desired effect in from two to three weeks, an operation (gastroenterostomy with pyloric occlusion) should be performed. The latter usually improves the prognosis.

(c) Duodenal ulcer accompanied by pyloric or duodenal stenosis.

Isochymia is here constantly present. In cases of beginning pyloric stenosis; duodenal alimentation and then stretching of the pylorus may be tried. The prognosis under this mode of treatment varies in different patients. The condition must be watched and the prognosis made accordingly. Should there be no improvement, or in case the stenosis is further advanced, so that the duodenal bucket fails to pass through the pylorus, an operation (gastroenterostomy) should be performed. Barring the dangers resulting from the surgical intervention, the result is here usually very good, and the prognosis accordingly favorable.

In duodenal stenosis, when situated below the papilla of Vater, there is bile constantly found in the stomach or in the vomitus. The treatment requires surgical intervention and the prognosis then becomes pretty good.

(d) Duodenal ulcer with periodically recurring hemorrhages.

In this group the main predominating symptom is a profuse hemorrhage (either hematemesis or melena or both), which periodically returns and endangers the life of the patient. An interval operation (gastroenterostomy eventually with pyloric occlusion) gives the best results and renders the prognosis more favorable. The latter must, however, be made with caution, as there may be a new hemorrhage even after apparent perfect recovery from the operation. In the latter event the prognosis becomes doubtful and worse with each repeated hemorrhage.

In groups (b), (c), and (d) the prognosis, in case no surgical intervention be undertaken, must not be made too favorable, as there is a possibility of perforation. With the gravity of symptoms the liability of this event increases. Continuous hypersecretion and severe pains are frequently prone to perforation. Appropriate measures should then be taken, in order to make the outlook brighter.

## PSEUDOLEUKEMIC ANEMIA OF VON JAKSCH

(Anemia Pseudoleukemica Infantum)

## CASE REPORT

By ROBERT KILDUFFE, JR.

Director, Pathological Laboratory, Chester Hospital; Bacteriologist, Borough of Marcus Hook; Visiting Physician, Medical Dispensary, Chester Hospital  
Chester, Pa.

In the classification of the anemias, the exact position to be accorded to the condition first described by Von Jaksch and Hayem under the name of anemia pseudoleukemica infantum still remains a matter of doubt.

In the present state of our knowledge of diseases of the blood, it is a debatable question as to whether or not it can be looked upon as a pathological entity, the weight of opinion being in favor of its being a secondary anemia generally associated with rachitis, tuberculosis or congenital syphilis. By some authorities it has been looked upon as an early stage of leukemia, a view hardly tenable if we consider the blood picture in detail and the usually favorable course of the disease; by others as a type of leukanemia, which term, however, has now been largely discarded as being incorrect in its significance, i.e., a combination of leukemia with the Addison-Biermer type of anemia, no such pathological condition having, as yet, ever been discovered.

The classification of the anemias is, as yet, in an unsettled and incomplete stage. If, however, we accept the main subdivisions of Morawitz<sup>1</sup> into (a) anemias due to temporary or more permanent increase in the amount of blood destruction, and (b) anemias due to decreased blood formation, Von Jaksch anemia is evidently placed among the former. A study of the blood picture readily sustains the view of a gradual, long-continued blood destruction, accompanied by more or less ineffectual efforts at blood regeneration.

Up to this point authorities are in accord; as to the cause of the gradual hemolysis much confusion exists. Supporting the theory of the possible influence of rachitis and other pathological conditions, may be mentioned Monti and Berggrun<sup>2</sup>, who reported twenty cases, sixteen of which were rachitic; Hunter<sup>3</sup>, Anders<sup>4</sup> and Barker<sup>5</sup>;

somewhat more non-committal as to the etiology, and expressing no definite opinion, are Simon<sup>6</sup> and Delafield<sup>7</sup>.

A somewhat cursory review of the earlier bibliography (vide Wentworth<sup>8</sup>) makes it evident that many of the cases reported as Von Jaksch anemia were simply cases of secondary anemia associated with varying degrees of splenic enlargement; and a casual review of later references reveals a paucity of case reports, and a somewhat abrupt dismissal of the subject, and it is because of this fact that the present case report is deemed of interest.

Undoubtedly rachitic changes are not infrequently associated with the blood condition; whether this is a coincidence or an etiologic factor remains to be settled. Certain it is, however, that, although rachitis, tuberculosis and congenital syphilis are by no means infrequent conditions in infancy, Von Jaksch anemia is a comparatively rare condition and one, it seems, not quite so easily explained, and requiring still further study.

Briefly, the condition may be described as a somewhat peculiar form of anemia occurring in children, usually in the first year, rarely in the second or third, characterized by a considerable increase in the number of leukocytes, enlargement of the spleen and liver, and often by hyperplasia of the lymph nodes.

The blood changes may be briefly summarized as follows:

1. Hemoglobin: decreased, but not as much as the red cells. Color index usually above 1.

2. Erythrocytes: markedly decreased; marked anisocytosis and poikilocytosis, polychromasia and basophilic degeneration. Nucleated reds—mainly normoblasts—are present in large numbers. (Chart II.)

3. Leukocytes: variable, though there is usually a leukocytosis of neutrophilic type of 20,000 or more (Chart I). This affords a ready means of differentiating the disease from pernicious anemia (Addison-Biermer type) and causes it to approach more closely to the acute hemolytic anemias. Myelocytes are often found (Chart II), thus forming the basis for an erroneous diagnosis of leukemia of myeloid type, if it is not remembered that, in children, the appearance of myelocytes in the circulating blood is a common occurrence in any condition in which the activity of the bone marrow is increased.

*Chart Showing Hemoglobin, Erythrocytic, and Leucocytic Curves*

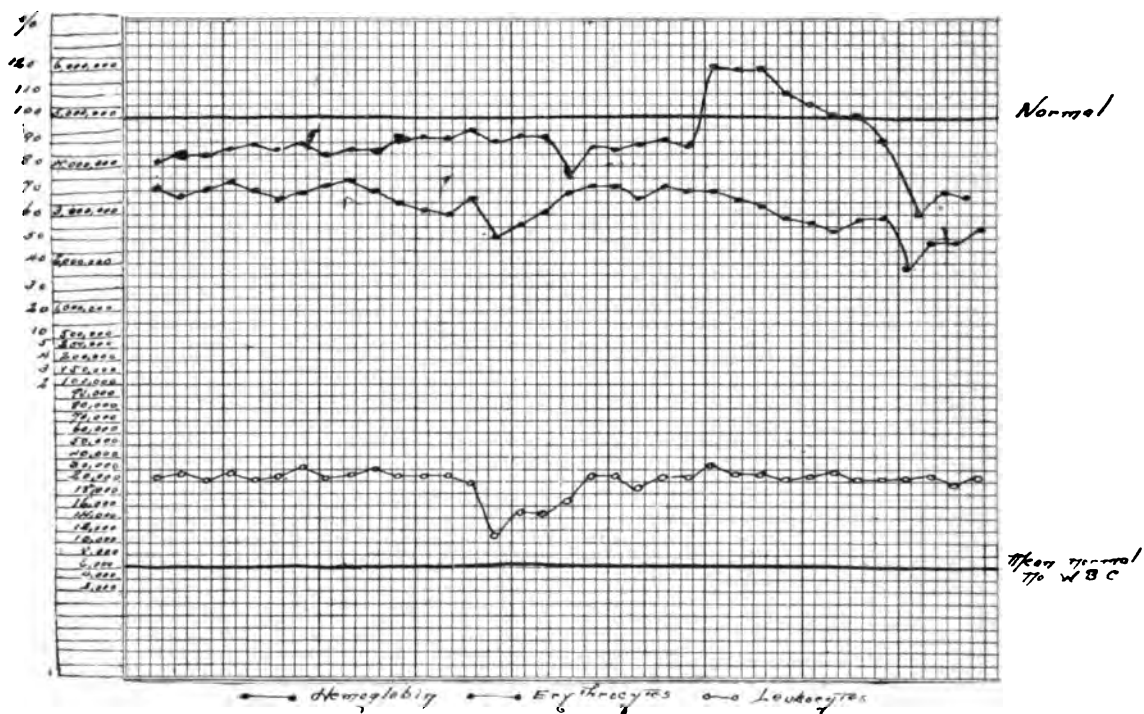


CHART I

*Chart Showing Erythroblast and Myelocyte Curves*

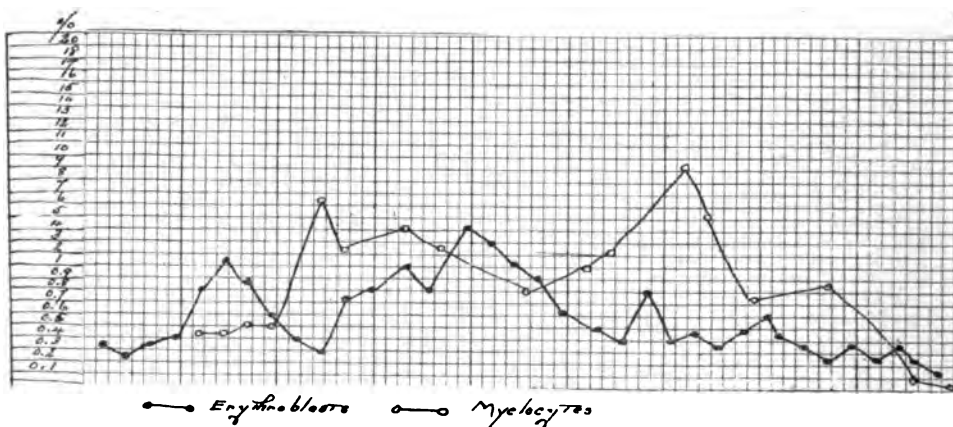


CHART II

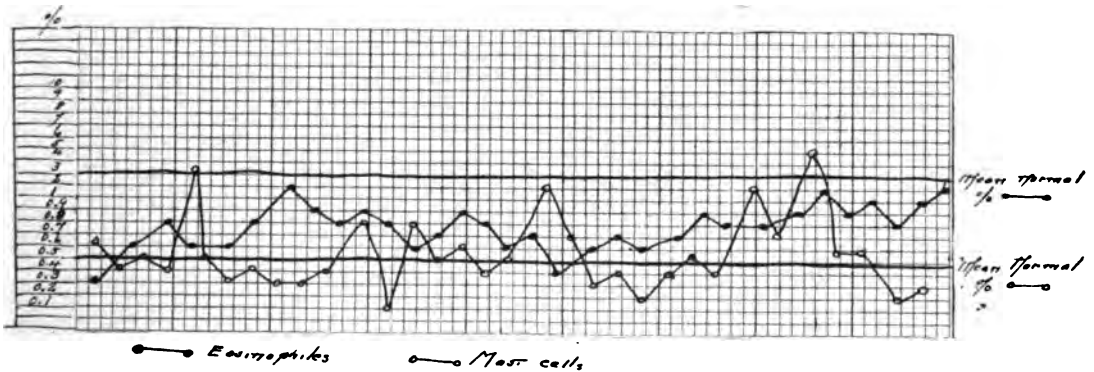
*Chart Showing Polymorphonuclear Curve*

CHART III

*Chart Showing Small Mononuclear Lymphocyte Curve  
and Large Mononuclear Lymphocyte Curve*

● Small Mononuclear Lymphocytes  
○ Large Mononuclear Lymphocytes

CHART IV

*Chart Showing Eosinophile and Mast Cell Cu.*

● Eosinophiles      ○ Mast cells

CHART V



Barker<sup>5</sup> says that the lymphocytes are occasionally absolutely increased. In the present case this was a prominent feature of the leukocytic picture (Chart IV).

The contention of Von Jaksch that all the different forms of leukocytes occur in their usual relative proportions is not borne out in the present case (see Charts II, IV and V).

4. Histological changes: in the main, the changes in the blood forming organs are similar to, but less prominent than, those of leukemia. Extensive hyperplasia of both erythropoietic and leukopoietic tissue of the bone marrow has been found, as well as myeloid metaplasia in the spleen, liver and lymph-glands. Typical leukemic changes are not found (Barker<sup>5</sup>).

Notes of the case herewith presented are as follows:

L. W., colored, aged 9 months, was admitted to the Chester Hospital 3/29/15 under the service of Dr. A. B. V. Orr, to whom thanks are due for permission to make this report.

Family history and past medical history, negative.

Status presents: for the past month and a half has had a slight cough associated with fretfulness and vomiting. Stools green, four to five daily. There has been some loss of weight and, for a short time previous to admission, the parents have noticed a gradual increase in the size of the abdomen. The family were of but moderate intelligence and no further history was obtainable.

Physical examination: Pulse and temperature normal at all times while under observation. Head square, suggesting the box shape, with wide fontanelles. Eyes, ears, nose and throat negative. Heart normal as to size, position and sounds. No abnormality of thyroid or thymus. Slight degree of enlargement of posterior cervical, axillary and inguinal glands. Over the upper lobe of the right lung, posteriorly, there is a slight dulness and a few crepitant râles; the chest is otherwise negative. The abdomen is quite distended, due partly to a slight degree of ascites, partly to a slight enlargement of the liver, and mainly to a conspicuous enlargement of the spleen, which is firm and hard and reaches almost to the umbilicus. The legs are extremely bowed and present evidences of hypertrophy of the epiphyseal discs.

A blood examination made on admission, by the interne, showed

white blood cells 24,000 with 53.3 per cent. of small lymphocytes. Von Pirquet negative. Urine not examined.

The diagnosis was in doubt for some time and was only revealed by systematic, complete examinations of the blood, which were continued until the child left the hospital, after three months, somewhat improved, when all trace of it was lost.

It will be noted that while, in the main, the picture presents much in common with that described as typical, there are several points of marked variance.

The erythrocytic curve was, in general, somewhat higher than usually described, and maintained a fairly constant level, varying from 4,320,000 to 4,020,000, with one high count of 6,210,000 and one low count of 3,610,000 per cubic millimeter.

Chart I illustrates the erythrocytic and hemoglobin curves.

The features of greatest interest lie in the pictures presented by the stained smears and differential counts. Anisocytosis, poikilocytosis, polychromatophilia and basophilic degeneration were marked and constantly present. Nucleated reds, mainly normoblasts, though megaloblasts were seen in varying numbers, were almost always present.

The leukocytic count (total) was also fairly constant, between 20 and 26,000, with one high count of 29,760 and one low count of 9,560. This is seen in Chart I. It is in the differential count that the most interesting features are seen. Prominent is the consistently high percentage of small mononuclear lymphocytes (Chart IV), which never fell below 38 per cent. and frequently rose higher. Neutrophils (Chart III) were very low at all times.

The variations in the nucleated reds and myelocytes is well shown in Chart II, and of interest, also, is the curve of the eosinophiles and mast cells, shown in Chart V.

The general picture is one of blood destruction, for which, it must be admitted, no satisfactory cause was found, and attempted repair, and corresponds closely to the condition described by Von Jaksch and which is still numbered among the diseases of the blood not yet thoroughly understood.

It would seem, however, that a condition dependent upon a pre-

vious or underlying rachitis, tuberculosis, or lues, should be more frequently encountered, and that the true explanation will comprise some other and, perhaps, distinct factor.

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### THE RELATIONSHIP OF THE DUCTLESS GLANDS TO ARTERIAL DISEASES

By JUDSON DALAND

Professor of Clinical Medicine, Graduate School of Medicine, University  
of Pennsylvania  
Philadelphia

Dr. Sajous, a pioneer in this field of work, has rendered a signal service in directing attention to the relationship of the ductless glands to arterial disease, and as a universally recognized authority on internal secretions, his opinions demand thoughtful consideration.

His communication is important because of the frequency and seriousness of diseases of the arteries, the alarming increase observed in recent years, the original views advanced as to the causation of arteriosclerosis by irregularities of the function of ductless glands, and the new measures advocated for prevention and treatment.

Arteriosclerosis is erroneously employed as a synonym for disease of the arteries, and leads to confused concepts of pathology. It is a subdivision of diseases of the arteries, and occurs as a *usual* or *occasional* result of *different* pathologic processes due to *different* causes. Atheroma due to age should be sharply differentiated.

Accumulated evidence secured at the bedside and by experimentation clearly proves that overfunctionating adrenals or thyroids produce arteriosclerosis, more especially if long continued or recurring with sufficient frequency. The exact manner in which

sclerosis is produced is open to question, and views of observers differ. The solution of the question is beset by many clinical and experimental difficulties, and is further complicated by the frequent association of multiple causes. Blood vessels vary congenitally. Some individuals possess unusual resistance to vascular disease, and a few show sclerosis as early as the fifth or seventh year without apparent cause. It is conceivable that these vessel wall changes are not primary and structural, but may be secondary, due to hyper-functionating ductless glands.

Long continued excessive muscular work causes arteriosclerosis, and although the supply of adrenalin is increased, this increase is a physiologic response to the needs of the musculature, and therefore *overwork* is the *primary* cause of the sclerosis. The big muscles of the blacksmith's right arm are occupational. Long continued excessive cerebral work causes sclerosis, more especially of the cerebral vessels, and here again the ductless glands are called upon for increased secretions, but the *primary* cause is excessive brain work.

The habitual ingestion of several quarts of water daily by a brickmaker caused advanced arteriosclerosis, a *part* of which was due to excessive labor. The transportation by the circulatory apparatus of a weight represented by so large a quantity of water adds greatly to the work of the cardiovascular system. Diabetes is often associated with the ingestion of large volumes of water, and in a similar manner produces arteriosclerosis, a *part* of which, however, is due to the products of altered metabolism, and an over- or under-activity of the thyroid or adrenals. Syphilis causes endarteritis, often obliterative, and aneurisms are common. The spirocheta pallida has been observed in the wall of the artery, and this organism and its toxins are the cause of the endarteritis. Rheumatism is accepted as a cause of arteriosclerosis. Recently evidence has been accumulated proving that rheumatism is in reality a secondary manifestation of a chronic septic focus, and is usually associated with one of the strains of streptococci, which has been observed in the wall of the artery. Whatever rôle the ductless glands play in this disease is secondary, and the toxic substances produced by the streptococci and by katabolism aid in the production of the vascular changes.

Gonorrhea, tuberculosis, pneumonia, and typhoid cause arterial disease, and their specific organisms have been found in the arterial lesion. When tuberculosis, pneumonia, or gonorrhea is associated with mixed infection, arterial disease is more likely to occur. The ductless glands in these diseases play a secondary or associated rôle.

Arteriosclerosis has followed scarlatina, variola, measles, influenza, typhoid and typhus fever. It is possible that the so-called rheumatism and the communicable diseases already mentioned stimulate the ductless glands, and secondarily produce the autolytic or adrenalin type of arteriosclerosis. On the other hand, it should not be forgotten that toxins made by pathogenic organisms may injure one or more coats of the artery, and so provide favorable conditions for their lodgment and growth.

Conflicting views exist as to the etiologic relationship of tea, coffee, alcohol and tobacco to arterial disease. Although Russians consume large quantities of tea, no effect was observed among the patients in many of the hospitals of Petrograd and Moscow.

Coffee exerts a marked influence over the cardiovascular system and in certain cases apparently causes arteriosclerosis, especially observable in Hungary, where this beverage is consumed in large quantities. Many cases of arteriosclerosis, supposed to be due to coffee, are neurasthenics, and the rôle of the ductless glands may therefore be larger than hitherto supposed.

Tobacco produces a marked influence over the cardiovascular and nervous systems, and when used in great excess, may also cause degeneration of the nerve endings. It is difficult to assign to tobacco its exact percentage of value *etiologically* in the production of arteriosclerosis, because it is almost constantly associated with the other causes of this disease.

A typical example occurred in three brothers, all using tobacco to great excess, leading to the supposition that it was the chief cause of the arteriosclerosis. They were markedly neurotic, living a strenuous business life, and at times indulging in excesses of food and wine. These patients doubtless also suffered from hypothyroidia and hypoadrenia.

The products of decomposition or fermentation of the intestinal contents produce arteriosclerosis, and may also stimulate the ductless glands.

French observers have long maintained this opinion, some believing that paracresol and indol of intestinal origin, due to the decomposition of nitrogenous materials, are able to evoke arterial disease.

All are agreed that gout causes arteriosclerosis. Although excess in food, especially rich in protein, with wine and insufficient exercise, are the chief causes, many attacks occur in the *absence* of these causes, apparently due to exhaustion or disturbances of the nervous system, well explained by Dr. Sajous' belief that katabolism may be induced by *lessening* of the activity of the adrenal center, with consequent diminution of the adreno-oxidase; and he furthermore believes that this explanation is equally true of lead poisoning.

From the foregoing, it is clear that in a number of instances the primary cause of arteriosclerosis may be *assisted* by hypoor hyperactivity of the thyroid and adrenals.

Arteriosclerosis and hypertension usually accompany kidney disease with renal inefficiency.

The retention of unknown toxic substances in the blood at first causes arterial spasm and later sclerosis, hitherto supposed to be *solely* due to the direct action of these toxic agents upon the vessel walls. It is probable that hyperfunctionating ductless glands assist this process.

Hypertension is a usual secondary manifestation of arteriosclerosis, and also occurs in many other diseases. When marked, it could *cause* or *increase* already existing arteriosclerosis by its physical effect, precisely as has been observed in sclerosis of the pulmonary arteries, with no sclerosis elsewhere, due to emphysema. Hypertension may occur as a sign of pre-sclerosis, or as a danger signal of advanced sclerosis.

With notable uniformity Graves' disease is associated with arterial walls that are degenerated and thickened and with increased blood pressure. It is believed that these changes are due primarily to hyperthyroidia and later to variations in this secretion. Additional unknown factors also probably exist, and there may be associated disease of the thymus.

Recent observations have clearly shown that anxiety, worry, fear, and what is usually understood by the term "strenuous living" cause hyperadrenia, which produces arteriosclerosis. *These causes of arterial disease are chiefly responsible, in the judgment of the served in recent years, the original views advanced as to the causa-*

*writer, for the remarkable increase in arterial disease in recent years.*

Dr. Sajous' classification by types serves a useful purpose, not only by detailing sequentially the mechanism by which internal secretions produce sclerosis of the arteries, but also by stimulating inquiry, observation, and experimentation. In order to emphasize my argument with this aspect of the problem, and to encourage discussion, the essential features of each type are briefly summarized:

An important feature of the autolytic type is the fact that trypsin, pancreatic in origin, is the ferment in the leukocyte which makes it a phagocyte, *able to destroy bacteria* in the intestinal tract, and after *migrating* causes *metabolism of proteins*, when in an alkaline medium, and *evokes katabolism* of toxic waste products, toxins and bacteria. Tissue cells contain zymogen or proferment of trypsin.

Proteins in excess sensitize this proferment, stimulate the thyroid and adrenals and so convert harmful surplusage of proteins in the tissue cells and blood into eliminable end-products. If now more protein be added, the stimulation of the thyroid and adrenals is increased, thereby increasing oxidation and causing fever, which activates the tissue cell proferment causing autolysis and *initiating arteriosclerosis*.

The adrenal type is characterized by hyperadrenia, causing constriction of the vaso vasorum of small or terminal arteries, which in turn causes lesions in the media and later in the other coats.

The denutritional type is characterized by hypothyroidia and hydroadrenia, causing defective oxidation and metabolism, which in turn causes arteriosclerosis, with low blood pressure. These conditions have been observed in myxedema, obesity, and advanced diabetes.

A study of the symptomatology of the three periods in the development of arteriosclerosis is of importance in order to diagnose the participation of the ductless glands in the process. Of the twenty-six signs and symptoms mentioned, almost half are common to many diseases. Hypertension, exaltation, nervousness, insomnia, pyrosis, hyperchlorhydria, venous engorgement, venous pulse, palpitation and congestion of the face, most clinicians would ascribe to disturbances of the nervous and cardiovascular systems,

and would *forget* the ductless glands. When all or a part of these symptoms are present and no other obvious explanation exists, they may be interpreted as the result of aberrations in the function of the adreno-thyroid apparatus. Pallor of the face, coldness of the extremities or inability to withstand cold may be *symptomatic* of vasomotor constriction, *adrenal* in origin.

The symptomatology of the autolytic and adrenal period of arteriosclerosis closely simulates that of neurasthenia, and this viewpoint opens a *new* field in the *therapy* of this group, care being first taken always *first* to *discover* and *remove* the cause.

It is desirable that a series of carefully observed cases presenting the symptoms of neurasthenia, be studied so as to determine whether organotherapy produces the desired results. A similar study should be made in the denutritional period, where definite results may be expected by alternately using and withholding adrenal, thyroid, or pancreatic extracts, the iodides, strophanthus, or digitalis. An intensive study of this character would give knowledge as to the frequency and variation of individual symptoms, and conditions under which each appears or disappears; and would eventually establish a *pathognomonic syndrome*. A great need exists for a technic by which the ductless gland apparatus may be accurately tested.

It has long been known that in hyperthyroidia, toxic symptoms are promptly induced by two or three grains of the extract of thyroid, and perhaps similar observations have been made in regard to hyperadrenia. Clinical observations of the effect of other substances should likewise be made, conjoined with a special study of the less characteristic symptoms.

The recognition of the participation of the ductless glands in the production of *symptoms* occurring in arteriosclerosis fundamentally modifies therapeusis and demands the use of old and well-tried remedies from this new point of view, as well as a more intelligent employment of organotherapy.

Practically, the *diagnosis* of the pre-sclerotic stage of arterial disease is *frequently impossible*, *always* difficult, and *usually* only probable. Therefore when arteriosclerosis is suspected or diagnosed, *prevention* or *arrest* may be accomplished by the prompt *detection* and *removal* of *one* or *more* of the causes of arterial



disease, coupling with this an estimate of the status of the ductless glands, in order to give the *maximum* aid to the patient in the *minimum* of time.

The greatness of Dr. Sajous' contribution consists in the principle that aberrations in the functions of the ductless glands, more especially of the thyroid and adrenals, *cause* arteriosclerosis. The acceptance of this principle adds *much* to our resources in the *prevention* and *treatment* of arterial disease, and encourages the expectation of a diminution in the morbidity and mortality of arteriosclerosis.

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### THE DUCTLESS GLANDS IN DEMENTIA PRAECOX

By FRANCIS X. DERCUM

Professor of Diseases of the Nervous System, Jefferson Medical College  
Philadelphia

Before entering into a consideration of the internal secretions in dementia praecox, it is important first to turn our attention to a number of facts of general but very great significance. Indeed, this is absolutely necessary in order that the changes in the glands of internal secretion and the possible rôle they play may be viewed in their proper perspective. We are impressed in the beginning with the large proportion of heredity in dementia praecox. In this all observers are agreed. Hereditary factors are variously estimated at from fifty-two per cent. by Schott to ninety per cent. by Zablocka. No doubt the differences in the percentages given by different observers are due to a divergence of view, first as to what should be included under hereditary factors, and secondly as to the affections which should be embraced by the general term dementia praecox. Kraepelin, at one time, found hereditary predisposition to mental diseases in seventy per cent. of his cases, though he thinks that this may possibly have been too high. He states that when the inquiry was limited to the direct heredity, i.e., to the occurrence of mental disease, suicide, or severe brain affections in the parents, it yielded 33.7 per cent., which he again regards as too low. From whatever point of view we approach the subject, however, the facts justify the general conclusion as to the frequency of neuropathic family histories in dementia praecox.

Such histories present not only instances of frank mental disease, but also of eccentric or unusual personalities, criminals, prostitutes, tramps, vagabonds and other degenerates. The wide range of the hereditary findings is also a fact of some significance. If the inquiry be limited to the direct transmission of dementia praecox, we find that such transmission is relatively infrequent, as a large number of cases, especially the hebephrenics and catatonics, never reach parenthood. It is otherwise, however, if we include the older, the paranoid cases. Ruedin from studies made of Kraepelin's material comes to the conclusion that dementia praecox is probably transmitted in accordance with the Mendelian law of heredity and appears as a recessive quality. He regards the marked predominance of the collateral and discontinuous inheritance over the direct inheritance, the increase of dementia praecox resulting from inbreeding and the numerical relation of those attacked to those remaining normal, as in favor of this view. The significance of a number of individuals of the same family suffering from dementia praecox cannot be questioned. I have personal knowledge of one family in whom no less than five individuals have suffered from this disease. It is also significant that Ruedin—in keeping with what has been already said—found in the families which he studied, other affections such as eccentric personalities and manic-depressive insanity. He also found that it was not at all infrequent for manic-depressive parents to produce children with dementia praecox, while the reverse—namely, manic-depressive children from dementia praecox parents—belonged to the rare exceptions. Of equal significance are such facts as the following: Ruedin noted that the late or last-born children of a family suffered more frequently from dementia praecox than the older children, and again, that immediately preceding or following the birth of a praecox patient there was frequently a history of miscarriage, premature birth or still-birth.

Other facts, the meaning of which is unmistakable, are those presented by the physical and psychic stigmata of deviation and arrest. Saiz places the frequency of the physical stigmata at seventy-five per cent. Among the latter are physical feebleness, retardation of growth, a too prolonged juvenile appearance, malformations or peculiarities of the shape of the skull, deep, narrow

and irregular palate, persistence of the intermaxillary bone, abnormalities of the ears, fingers or toes, imperfections and anomalies of the teeth and other morphological peculiarities.

It may be correct to accept Ruedin's inferences as to the hereditary transmission of dementia praecox as a recessive quality in accordance with Mendelian law. However, the foregoing facts suggest that in addition, the germ plasm may suffer from impairments that affect its general morphological and biological character and profoundly lower its possibilities of growth and development. Among causes which may thus grossly impair the germ plasm we have reason to believe are infections and intoxications affecting the parent. Pilcz, Klutscheff and others have published suggestive statistics as to the frequency of syphilis in the parents, while Diem, Fuhrmann, Ruedin, Wolfsohn and others have published studies on alcoholism in the parents alike suggestive and significant. That syphilis plays a rôle in a not inconsiderable number of cases is proven by the frequency of the Wassermann reaction in the patients themselves. Bahr, for instance, found it in so large a proportion as 32.1 per cent. Such facts do not mean that the patients are necessarily suffering from a syphilitic disease of the nervous system, but rather that the organism as a whole has been hampered, made deviate and degenerate in its development by the presence of the spirochete and its toxins, i.e., that the development of the organism as a whole—and included in this the development of the glands of internal secretion—has been so inhibited and altered that, at a given point of its life, the organism breaks down by reason of an abnormal and toxic metabolism. Again, it is not necessary that the Wassermann or other tests should yield a positive result. It suffices if the infection has damaged the germ plasm of the parent, and, in keeping with this is the fact that clinical evidences of inherited syphilis are absent in the great mass of cases. Finally, that alcohol damages the germ plasm of the parent must, I think, be freely conceded and its discussion need not detain us here.

The question whether other poisons and infections also play a rôle in causing damage to the germ plasm cannot be definitely answered; but such action is neither impossible nor improbable. In any event, however, their action must be vastly less important than that of syphilis or alcohol.

It may be possible, let us repeat, that the germ plasm may be laden with a direct tendency to the development of dementia praecox, and which tendency is transmitted as a recessive quality, but that the germ plasm may also suffer gross impairments, the results of syphilis and alcohol and perhaps other infections and intoxications must, I think, be frankly admitted. Further, in keeping with this view is the fact that dementia praecox presents itself not as a specific, a sharply delimited clinical entity, but as a group of mental affections which possess the one common factor of endogenous deterioration.

The above considerations point clearly to the involvement of the organism as a whole. We should remember, too, that the existence of the various evidences of morphological deviation visible to clinical observation also imply that other and perhaps more fundamental deviations are present in the organism throughout. Such an organism must present not only abnormalities of structure, but also abnormalities of function and especially of metabolism. That the internal secretions play a rôle in the general disturbance is extremely probable. Dr. Sajous has pointed out the cogent facts indicating an important rôle played by the thymus gland. In keeping with the view he has presented are not only the facts pointing to a defective nervous development, but also the observations of Barbo and Haberkandl of the occurrence of osteomalacia in dementia praecox. However, I believe that we should be very careful in drawing our conclusions. Various facts point to other structures as well. Thus, that the thyroid gland may present abnormalities is a matter of common knowledge. Occasionally it is enlarged, more frequently, in my own experience, it is small. Thus, in seven of my own autopsies in which the thyroid gland was weighed, five were little more than half the normal weight, one was one-fourth the normal and only one approximated the normal. Again, out of eight pairs of adrenal glands five were greatly in excess, one decidedly below normal and two about normal. Such facts as these are, of course, impossible of detailed explanation; they merely point to a disturbance of the internal secretions. Changes in the glands of internal secretion were also found by Farrant. By far the most detailed study of the weight of the ductless glands in the insane is that made by Kojima in the pathological laboratory of the Clay-

bury Asylum. As far as can be gathered from his tables, his results are practically in accord with my own, for the cases labeled insanities of adolescence and dementia praecox.

The thymus, pituitary and parathyroid glands studied by Ellis and myself did not reveal changes capable of interpretation, though here and there the findings suggested pathological conditions. Of our thyroid glands three out of the seven showed changes in the colloid material and four regressive changes in the acinar cells. The most constant findings in the adrenal picture was the small amount of fat in the cells of the cortex; possibly this indicated a lessened functional activity. However, that there are other glands which probably play a rôle in dementia praecox, the evidence strongly indicates. Clinically our attention is strongly attracted to the sex-glands. We are confronted by the anomalies of menstruation, or by the delayed and imperfect establishment of puberty, on the one hand, or of sexual precocity on the other. Again, there is the history of sexual excesses, sexual vagaries and perversions. A relation to the sex glands is further indicated by the accentuation of symptoms often observed during a menstrual epoch and by the fact that dementia praecox now and then has its incidence in a pregnancy or in repeated pregnancies or in a miscarriage, as though sex gland exhaustion played a rôle. Various writers, among them Tsisch, Lomer and Kraepelin, have assigned an importance to the sex glands. Lomer, particularly, indicated a disturbance of the internal secretion of the latter, but it remained for Fauser to throw an especially illuminating light on the subject. Fauser, as is doubtless well known to my hearers, found in the serum of dementia praecox cases, defensive ferments against the sex glands and against the cortex. It would appear from Fauser's investigations that in dementia praecox a primary dysfunction of the sex glands leads to the entrance into the blood of unchanged sex gland protein, and that in the subsequent breaking up of this protein, substances are formed which are injurious to the cortex, and which bring about the destruction, the lysis, of the latter. The substance which enters the circulation is the internal secretion, the hormone of the sex gland, not, of course, the germinal product. The blood of male dementia praecox cases digests testicle only, not ovary; that of female cases, ovary only, not testicle. Fauser's results have

been confirmed by a large number of investigators, among them, Roemer, Bundschuh, Kafka, Ahrens, W. Mayer, Neue, J. Fischer, and many others. Only in dementia praecox cases could Fauser demonstrate defensive ferments against the sex glands. In cases in which a digestion of the sex glands was unexpectedly found, and in which a diagnosis had previously been made of manic-depressive insanity or of other functional psychoses, the further clinical course of the cases proved that they were really cases of dementia praecox and that the serologic diagnosis had been the correct one. In a number of less definite cases of dementia praecox in which defensive ferments against the sex glands were found only at times, it seemed as though this dysfunction ran parallel with fluctuations in the clinical course. In some cases, again, in a terminal and stationary condition; that is, in cases in which the pathologic process had run its course, no defensive ferments were found against either the sex glands or cortex. Not infrequently, defensive ferments were also found against the thyroid and against the adrenals. Ludlum, of Philadelphia, has also obtained positive results in the thymus. In the cases which reacted to thymus the patients were small, light of build, and presented morphological features of arrest and other abnormalities. He regards them as cases of under-activity of the thymus. Whatever the future may reveal, there appears to be no escape from the conclusion that in dementia praecox there is a deranged metabolism, an autotoxic state, in which abnormalities of the internal secretions play a leading rôle.

It is very probable, further, that in dementia praecox toxic action is not limited to the cortex. Many of the symptoms suggest the action of toxins upon the sympathetic and autonomic nervous systems as well. How the latter react to poisons, e.g., the various alkaloids, atropin, pilocarpin, morphin, etc., is more or less definitely known. Our knowledge likewise extends to the action of the internal secretions, though here, as a matter of fact, our knowledge is less complete. Much information is, however, in our possession. Adrenalin, for instance, induces contraction of the blood vessels, acceleration of the heart's action, dilatation of the pupils, drying of the mucous membranes, lessening of the secretion of the salivary glands and lessening of the motility and secretions of the stomach and intestines. Similarly the action of the thyroid and

pituitary secretions are attended by phenomena that can only be explained by their action upon the autonomic or sympathetic systems. For instance, how full doses of thyroid preparations accelerate the heart's action, increase the secretion from the skin and of the intestinal tract and increase peristalsis, and how markedly pituitary preparations influence the rate of the heart, blood pressure and other processes is also well known. That some of the phenomena observed in cases of dementia praecox are referable to mere quantitative increase or decrease of the various internal secretions, and that others still are due to perversions of these secretions is extremely probable. That phenomena pointing to the action of toxins on the sympathetic and autonomic nervous systems are present in dementia praecox cannot be denied. There are the phenomena presented by the circulatory apparatus, the digestive tract and such other very special forms of apparatus as the iris. In the recognition of the facts lies the explanation of many of the symptoms. Among the latter are the atonic indigestion, the constipation and the dryness of the digestive tract, the phenomena presented by the circulation, the alterations of cardiac rhythm, the fall of blood pressure, the lividity, dryness, moisture, or other conditions of the body surface, the dilatation or other anomalies of the pupil; and other symptoms as well. The point which should be emphasized is that these phenomena must be referred to a toxic action, an action which expresses itself through the autonomic or sympathetic nervous systems. The last mentioned fact acquires additional significance in our studies in mental disease, when we reflect that it is through this system that the emotions, the affects, mainly find physical expression.

It would appear that in dementia praecox the various glands of internal secretion have suffered in the course of the development of the organism, so that their respective functions are subsequently imperfectly and aberrantly performed. It is not at all unlikely that while a number of glands—perhaps the entire chain—are involved in most cases, certain glands by their action, e.g., the sex glands, may dominate the picture; in others again it is the thymus; in still others it is the system of the pituitary, thyroid and adrenals. In favor of the special rôle played by the thymus is perhaps the fact that cases of dementia praecox frequently betray in childhood the

forerunners of the affection. Kraepelin states that very frequently, especially in the case of male patients, it was brought to light that as children the patients had been markedly quiet, shy, retiring, had formed no friendships, had lived only for themselves. Again, especially in girls, accounts were received of irritability, nervousness, stubbornness and obstinacy. Then, again, a smaller group was noteworthy, mostly boys, in which the children were lazy, disliked work, were unsteady, prone to misdemeanors, held to nothing and finally became tramps and vagabonds. In contrast to these are others, likewise more frequently met with in boys, who as children are characterized by docility, good nature, great conscientiousness and diligence, are unusually good and who hold themselves aloof from all improper conduct. It is not difficult to correlate the reserve and obstinacy with the later appearing symptom of negativism; the oddities and eccentricities with the subsequent symptom of impulsivity, and the docility, on the other hand, with the subsequent symptom of automatism at command. It would appear, indeed, that frequently the character, demeanor and conduct of the child foreshadow the later appearing symptoms of the dementia praecox and that the affection really has its inception in childhood.

It should be added in concluding that it cannot be inferred from Kojima's studies that the changes found in the glands of internal secretion, e.g., the abnormalities in their weights, are characteristic for any form of mental disease. However, it must be remembered that Kojima did not study the thymus.

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## THE FIELD OF INTERNAL MEDICINE\*

By REYNOLD WEBB WILCOX

President of the American College of Physicians

New York

It is an occasion of more than ordinary importance when the American Congress on Internal Medicine convenes for its second annual meeting, which is, however, its first scientific session. It has completed its physical organization and now presents its scien-

\*Presidential address, delivered before the American Congress on Internal Medicine, at Hotel Astor, New York City, on December 28, 1916.



tific programme. Its organization marks a new era in American medicine, and the programme which has been chosen determines a new standard for scientific work in the profession of which we are the exponents.

The organization of this Congress does not signify the differentiation of a new specialty, but the delimitation of the oldest branch of the healing art, for it is probable that disease received earlier attention than injury. Whatever may be the fact, it is, however, true that medicine and surgery were yet undifferentiated in practice throughout the era of the prehistoric man, and even for many centuries thereafter. As war became more and more an organized operation and campaigns were planned, the care of the wounded devolved upon the practitioner of the healing art, and surgery became differentiated in name as well as practice, and the chief surgeon of the army was often the physician of the ruling prince or king. Nor, indeed, did his professional title always change, for even so late as the War of the Revolution in this country the title of the medical officer was physician and not surgeon. Yet today in the army the title of surgeon prevails, while the more important work of the military practitioner, whether considered from the combatant or the altruistic standpoint, is medical rather than surgical.

What then is the domain of internal medicine? Shall we define it as what remains after surgery and the narrower specialties, as ophthalmology, otology, laryngology, gynecology, andrology and urology, or whatever of it belongs to the preceding two categories, are subtracted? Or shall we still further diminish its field by eliminating neurology, psychiatry, pediatrics and dermatology? The position of the dermatologist calls for especial consideration. It is conceded that surgery does not claim him. If we follow the Vienna school in assuming that the skin is an organ, as the eye or the ear, he would be an exponent of one of the narrower fields of specialism. If we should adhere to the tenets of the London school and expect the attention to be directed to the study of systemic conditions, which that school has emphasized, he could readily be enrolled as a practitioner of internal medicine. In fact, one of the greatest names of that department of the healing art was Hutchinson, whose fame rests largely upon a disease, syphilis, which is clearly in the field of internal medicine. If we are influenced

by the Paris school, our decision must rest somewhat in doubt. However, this is a question upon which the Congress eventually must take official action. A definition which is predicated solely upon exclusion is neither logical nor final. The schismatic operations being repeated, the remaining moiety might readily become negligible. A definition must be not only inclusive, but as well exclusive. We may define the domain of internal medicine as including:

1. Diseases caused by parasites: Psorospermiasis, distomiasis, trypanosomiasis and by nematodes, cestodes and parasitic insects from arachnidæ to pediculi, either as directly causing disease or by their acting as carriers.

2. Infectious diseases, of which enteric fever, diphtheria, infectious pneumonia, tuberculosis, erysipelas, syphilis and the eruptive fevers, communicable or contagious, represent various types. These number nearly ninety, the majority of definite and known causation, all readily recognizable, and all presenting pathological manifestations of which the treatment must fall to the lot of the internist.

3. Constitutional diseases, such as gout, diabetes, scurvy, rickets and others.

4. Intoxications, including the various metallic poisonings, alcoholism and other drug poisonings, food and occupational poisonings, and the results of exposure to high temperatures.

5. Diseases of the digestive system and its adnexa, the liver, and pancreas.

6. Diseases of the blood and of the ductless glands, which are not only of increasing interest and importance, but are likely, in the future, to necessitate a new classification.

7. Diseases of the circulatory system: heart, pericardium and blood vessels.

8. Diseases of the respiratory system, including those of the pleura.

9. Diseases of the mediastinum, few in number, and relatively rare, but of enormous difficulty in diagnosis.

10. Diseases of the urinary system.

11. Diseases of the nervous system, including those of the mind.

12. Diseases of the muscular system; the myosites, the dys-

trophies and the disorders of function of which myasthenia, myotonia and paramyoclonus are types.

It cannot be assumed that this classification is final, because not only are individual diseases constantly changing from one division to another, as, for instance, pneumonia from diseases of the respiratory system to the infectious diseases, but also some groups may be merged together as our knowledge of etiology increases. The terrain will remain the same, although the boundaries of the different divisions may change.

This, then, is the broad domain of internal medicine; which is of such eminent importance in the life-history of mankind, and which dominates all the limited specialties of the healing art. Its successful cultivation demands that all sciences render aid—physics, and its younger brother, physical chemistry, botany, zoölogy and especially biology in the broader acceptance. Upon this advanced knowledge and the logical interpretations of it, and the legitimate applications to that complex category of physico-chemical relationships, which we call life, must depend substantial and beneficial progress in internal medicine. In its domain are to be found the greatest incidence of disease, either in number or importance of disability and the preponderating causes of death.

Can this field of internal medicine be divided? There is no doubt that in practice this may be done to a limited extent. For instance, diseases of the nervous system can be separated from internal medicine and the neurologist may confine his efforts to diseases of that system, and further the psychiatrist may limit his practice to the diseases of the mind, but he will be the more useful alienist whose knowledge of diseases of the nervous system is the greater, and both will be more completely experts the more accurate and comprehensive their knowledge of the broad field of internal medicine has become. The medical diseases of infancy and childhood have many exponents who properly confine their practice to those periods, because not only does the physiology and pathology of the constructive period of the body differ from that of the adult, but diagnosis and treatment present variant problems. So also do the diseases incident to old age, but with a solution hopeless as to the final outcome, though fruitful in alleviating many of its discomforts. In passing, attention might be called to the fact that

the time during which the pediatricist exercises his functions does not always end with puberty, but may even extend itself through that of childhood, which some of our pedagogues, notably college presidents, assume to continue during the entire period of education which is necessary to adapt the human being to his environment and to fit him for his greatest usefulness, and this period has been mentioned as thirty years. When we reflect that the storm and stress of modern civilization have shortened the span of human life, and men may be octogenarians in body, if not mind, at sixty or even earlier, the period of adult life may become a brief one, and the pediatricist and the geriatrist may almost meet in their separate fields of activity. Thus it is evident that both should be thoroughly versed in the domain of internal medicine. It is indeed true that the foundation of senility is laid in the period of childhood, and that man usually begins to die the moment that he is born. The laboratory workers, whether in the field of biological chemistry, bacteriology, parasitology, physiological therapeutics and in physics, especially in electro-therapeutics and röntgenology, certainly have claim upon our consideration. To them internal medicine owes much, not only in indicating new avenues of progress, but as well in scientific demonstration of the verity of what empirically we have established as facts in internal medicine, and we have made but a beginning.

Granting that the foregoing are legitimate subdivisions of scientific endeavor and practical realization in the field of internal medicine, the question of further limitation of practice immediately suggests itself. The natural cleavage would be in accordance with the classification which has been given at the outset, according to the different physiological systems. The diseases of the circulatory system depend upon a distinct group of organs, but the results of imperfect function or structural disease are as far reaching as the circulation of the blood, and their symptomatology may be the symptoms ascribable to any one, many, or all of the physiological systems. The worker in the field of diseases of the circulatory system may limit his practice, but he will be expert only as he is versed in internal medicine. The stomach specialist *per se* has no reason for his existence. If he devotes himself to diseases of the digestive system and includes with these the disorders of metabolism

and the constitutional diseases, his field is broad enough to occupy his best endeavors, but here again he must be conversant with the established facts of the larger territory occupied by internal medicine if he shall attain real usefulness.

So might be cited the group of infectious diseases, often presenting problems of diagnosis, and the same statement applies as to the importance of a broad and comprehensive knowledge of internal medicine. Further than this, the exponent of internal medicine, no matter whether he shall be the one considering the field in its entirety, or one limiting his work to a subdivision of it, must know syphilis in all its manifestations, and its results. For its widely spread incidence must always be taken into account as modifying disease or dominating therapy throughout the whole field of internal medicine. Its signs and symptoms are often so bizarre that even its recognition, at times, is exceedingly difficult, the Wassermann reaction notwithstanding. The range and scope of internal medicine are well defined and its domain is accurately marked out, both inclusively and exclusively.

The relationship of surgery to internal medicine is intimate, and yet the differentiation is apparent. There is hardly a disease in the entire range of internal medicine but that at some time in its course, or in the presence of some complication, surgical intervention may be called for. And this intervention may be necessary at an early date—in fact, so soon as the diagnosis can be established, as, for instance, in acute appendicitis. It is well to bear in mind that what are often denominated border-line diseases are really those in which the activities of the medical attention and surgical practice are concurrent. There are others, for example, cholelithiasis, in which the etiology falls in the domain of internal medicine, the important item of relief comes under the jurisdiction of the surgeon, while the final cure comes within the purview of internal medicine.

As internal medicine and its contributing laboratory work has laid the foundations for real surgical advance, so internal medicine can make surgery of its highest possible value. The best surgical work done in this country to-day is accomplished by intimate scientific coöperation between the skilled exponents of internal medicine and the expert operator. The mere operator may be an agent of

destruction, no matter how deft he may be or how perfect his technic. He only reaches his highest usefulness when he has a broad knowledge of internal medicine; that is, becomes a surgeon, or relies upon other experts for diagnosis and an analysis of the real condition of the organs and functions of the patient. Naturally, this statement does not apply in its entirety to operations of urgency. When the surgeon attempts to be a general practitioner we are likely to be informed as to the surgery of dyspepsia or the ablation of a function. Medical surgery is not likely to yield the best results and surgical medicine is sure to be disappointing.

The relationship of internal medicine to surgery is fundamental and necessary—this fact must be recognized by the practitioners of both—but it must be founded on mutual confidence and respect for technical skill. Although internal medicine dominates the situation, it does not detract from a just admiration for the wonderful results which modern surgery has accomplished. The mechanical skill and the perfect technic of the operator are rewarded by appreciation, but the intellectual work of the trained exponent of internal medicine is equally worthy of admiration.

We have defined the field of internal medicine and have shown its relationship to the coördinate branch of the healing art—surgery—and the narrow specialties, and now we must define our name. It is a curious fact that the practitioners of internal medicine have not yet, by common consent, so far as this country is concerned, received a distinctive name. The term “diagnostician” has been suggested. Diagnosticians we certainly are, and we are proud to be considered as such, but we realize, better probably than any other group of practitioners, that diagnosis is not the sum total of our efforts, but only the conclusion of the first stage of our work, and merely preliminary to the part that is most important to our patients, which is treatment. We certainly are not general practitioners, either in theory or practice. For, with the mass of accumulated facts and the logical deductions therefrom, neither the learning of an Aristotle nor the intellect of a Bacon, nor both combined, if such a genius were possible, could result in so broad a knowledge, so vast an experience, and so great a technical skill that all phases of scientific endeavor could be marked with such a degree of usefulness as we believe adequate for professional work.

Nor does this statement conflict with the opinion that specialists, both broad and narrow, are better specialists if the earlier years of their career are devoted to general practice, and the broader their knowledge and the larger their experience in the general field the more likely are they to become really expert in the smaller field to which their natural aptitude or especial opportunities may have limited them. The name "internist" is undoubtedly the proper one for those whose activities are circumscribed by the limits which have been set down earlier in this address. The term "physician" too often is assumed to have the qualifying adjective "general" omitted, and is not distinctive. In the profession, even, one who has worked exclusively in the field of internal medicine for a quarter of a century, eschewing surgery, obstetrics and the narrower specialties, who has been a teacher of medicine and an author of text-books upon its practice, is frequently and erroneously designated as a "general practitioner." In Great Britain we are known as "internists"; on the Continent "internal medicine" is recognized; let us be known in this country as internists, and be willing to define the term until such time as the profession and the people know what it means, and medical associations, big and little, representing or not medical science, afford the designation official recognition. We must teach that the "internist" is an educated and trained physician, who gives his best endeavors to an accurately delimited field, known as "internal medicine," and that the real internist is not only a specialist, but, what is far more rare, an expert. It is to the internist that the heritage of the earlier physicians has come. This is the American Congress on Internal Medicine, and we are the descendants of men who have served their time and generation, and have left their impress upon American medicine.

We probably recall John Morgan (b. 1736), of Philadelphia, pioneer with William Shippen, Jr. (b. 1736), in the establishment of a school for medical education, the grandfather of American medicine, and Samuel Bard (b. 1742), of New York, who was identified with the earliest medical instruction in this city. Probably the best known name of this generation, but whose activities were so varied that he is better known in connection with the revolutionary period, was the Father of American Medicine, Benjamin

Rush (b. 1745), of Philadelphia; his third edition of "Medical Inquiries and Observations Upon the Diseases of the Mind" (his first was in 1812) lies before me bearing the date 1827. It is interesting, if not instructive, and written in idiomatic and classical English well worthy of Addison, it is a model for the medical editor of today.

Another worthy of this period was Nathan Smith (b. 1762), who at Dartmouth did not occupy a professional chair, but rather an entire settee, for he taught medicine, surgery, anatomy, therapeutics, botany, physiology and chemistry. A contemporary of Daniel Webster, although there was twenty years difference in their ages, he contributed much to medical science, as well as establishing two medical schools. In 1813 he came to New Haven and repeated his pioneer work in founding the medical school which subsequently became a part of Yale. He died in 1829, and his grave in the Grove Street cemetery is still a Mecca for medical men. As professor of the theory and practice of physics and surgery, his name is upon my grandfather's diploma, in 1819.

The fourth to establish medical schools was Benjamin Waterhouse, the physician, who with John Warren (b. 1753), made possible a medical school in connection with Harvard, in 1782. He also was the first to introduce Jennerian vaccination into this country, which he did in 1800. In all the early efforts to establish medical instruction in this country, the medical aspect of the healing art looms large.

The son of Nathan Smith, Nathan R. Smith, in 1825 published his "Physiological Essay on Digestion," which antedated much that was subsequently discovered. My copy from my grandfather's library bears upon its title page this sentence: "It is no small part of science to be well acquainted with its real boundaries; but it is necessary also to know what it is which truly exists within these boundaries, and what it is which is only fabled to exist." A little later than this time we recall William Beaumont (b. 1784), pioneer physiologist of this country, whose experiments and observations on the "Gastric Juice and Physiology of Digestion," Plattsburg, 1833, were epoch making. Curiously enough, the place of his observations upon Alexis St. Martin was the battlefield of 1814, a



portion of which is now occupied by the Military Training Camp, with which you are all familiar.

Passing by many contemporary lesser lights, we come to another epoch-making medical advance. While we may speak of the work of Crawford W. Long and his rival claimants to priority, Jackson, Wells and Marcy, this fact is firmly established: It was William T. G. Morton (b. 1819) who first publicly demonstrated that by the inhalation of ether unconsciousness sufficiently profound to permit of surgical interference could be produced by medical means. This was on October 16, 1846; the place was the Massachusetts General Hospital, and there are those present in this room who have heard the narration of that event from the lips of those present at the demonstration. It was also another graduate of Harvard, Oliver Wendell Holmes, in his early days a physician, but better known as an author of delightful fiction, both prose and poetry, and a teacher of anatomy, who coined the word "anesthesia," by which this priceless boon to humanity is known throughout the whole world.

Medicine has made modern surgery possible, and to it credit must be given for the wonderful surgical work that is being done today. Parenthetically it might be remarked that anesthesia has also permitted some very mediocre surgery—thus not every great blessing is entirely unalloyed.

Fundamental, also, to the present value of surgery is antisepsis, which has been developed as purely a medical problem, and which has led to asepsis as an ideal of more or less complete realization, although the present European conflict which is now raging has demonstrated that chemical antisepsis is still of great importance.

An epoch-making book of a later date was "Nature in Disease," by Jacob Bigelow (b. 1787), a Harvard professor; my copy, the second edition, bears the date of 1859, although the first was published in 1854. His views on self-limited diseases directed rigid analysis of the value of therapeutic measures. In his dedication of it to Robley Dunglison (b. 1798), the medical lexicographer and another of our medical Nestors, we find: "I am sure that you will unite with me in admitting that the experience of a long professional life is the best corrective of the exaggerated estimate which we are liable to form, or imbibe, in our earlier years, as

to the power of medication to control disease." Dunglison also occupied a settee at the University of Maryland, for he was professor of materia medica, therapeutics, hygiene and medical jurisprudence, and his writings comprised systematic treatises upon "Physiology," "Hygiene," "Therapeutics," "Practice" and "Materia Medica."

W. W. Gerhard (b. 1809), of Philadelphia, must claim our attention for a moment, for he was the "first man to distinguish clearly the difference between typhus and typhoid fevers."

Of the more recent developments in medical science due to American medicine we need only to allude to our antityphoid vaccine, the most efficient of any country, to our work on uncinariasis, yellow-fever, pellagra and malaria, to our work on sanitation in tropical and subtropical countries, which has given such brilliant results, the names of those who have made medical history are upon our lips, some are yet living—others, alas, have fallen, martyrs to medical science.

The greatest triumphs over diseases, and even death, achieved during the last half century have been medical rather than surgical. They have been the discovery of the *causa causans* of disease, and the separation of the infections, due to bacteria, or protozoa or other organisms of the lower zoölogical orders, from the inflammations and degenerations. The direct result of our knowledge of etiology has resulted in the preventing of the incidence of disease on the one hand, by intelligent hygiene, and by extensions of the theory of Jennerian vaccination to other diseases, notably diphtheria, tetanus and enteric fever. And a further direct result has been the ability to cure such infections during brief periods, as has been particularly demonstrated in diphtheria, malaria and syphilis, and not only this, but as well, by serological methods, to demonstrate that the cure is absolute and permanent. In others, as acute rheumatic polyarthritis, we have found methods to markedly shorten its duration, directly alleviate suffering, and prevent frequent complications.

Among the constitutional diseases absolute prevention and relative cure has been brought about in some, for example, in scorbutus, diabetes and gout, with a minimizing of suffering in some and averting a fatal issue in others. In diseases of the circulatory

system medicine has made startling advances in drug therapy and physical procedures, so that no longer are the problems approached with other than confident expectation of benefit and relative cure so long as degenerations can be checked in their course and structural changes have not extended beyond the possibility of functional recovery. The same may be said of the diseases of the respiratory, digestive and urinary systems. Among the great triumphs of recent medicine may be cited the accurate and productive studies upon the blood, and work upon the functions of the ductless glands, the results of which have been far reaching and of inestimable value, and whose importance in health and disease cannot be overestimated. I need not remind you that these problems are purely the problems of internal medicine, and their solution depends solely upon the internist. In fine, the most important developments in the healing art, important not only in the larger number of diseases and of major importance, but as well in the larger number of individuals afflicted, has been in the domain of internal medicine. And, furthermore, the greatest progress in the immediate future must of necessity be in this very field.

The record of the distinguished physicians, our medical ancestors, is far too long to be adequately presented in a ponderous tome, let alone in an address. They were giants in those days, of thorough mental training and discipline, of accurate and painstaking observation, of rigid logical analysis and productive clinical deductions. They have contributed in large measure to the advance of medical science and therapeutic art. We are the legatees of these physicians of a magnificent medical past, and as internists we are the trustees of the glorious internal medicine of the future, whose soundness in scientific basis, whose development in the alleviation of suffering and the prevention and cure of disease, and whose value to suffering humanity only the seer must venture to predict. This is the function of the Congress on Internal Medicine; to view with reverence the foundations laid down, broad and deep by the physicians, our medical ancestors, and as internists to raise upon them a useful structure for the healing of the nations.

The Congress on Internal Medicine has for its *raison d'être* (1) to define its domain, (2) to procure recognition of the designation "internists," (3) to promote solidarity of the interest and achieve-

ment among them and (4), finally and most important, to advance the science of biological medicine, of which we are the exponents: (a) by the selection of experts who shall report the results of their investigations of important problems and of intensive clinical study and experience, (b) by extending the sphere of influence of the constructive workers in internal medicine through publication of their conclusions, (c) by authoritatively instructing the public in regard to the great problems of health through the official departments and services now organized, and thus render them more efficient.

I do not approach this constructive work of building up the American Congress on Internal Medicine with any misgivings as to the result, even when one considers the ambitious programme which is outlined.

We believe that the time has come when the internists shall be united for scientific advancement, and for the benefit of suffering humanity, and that this organization shall be controlled by those who have been instrumental in developing internal medicine by modern scientific methods, and that this does not meet a temporary need alone, but its existence and importance will reach far into the future. Of its permanent success I have not the slightest misgivings.

The American College of Physicians, through its council, admits to its fellowship, by election, those recommended by the council of the American Congress on Internal Medicine from among its members. The membership in the college is restricted to those whose practice is generally in the field of internal medicine or especially in the recognized departments of the same. Its obligations are those of a gentleman and a member of a learned profession. It has been said that the American College of Physicians creates an aristocracy among the internists. The observer has discerned the purpose of the founders of the congress and of the college. He forgets that the graduate who has earned the bachelor of arts degree has become a member of the aristocracy of letters created centuries ago. He also forgets that the master of arts degree, won after study and examinations, admits the bachelor of arts to a smaller group in that aristocracy of letters. These are honors

which mark attainment of the individual in his progress toward appreciation of the higher relationships of life.

The degree which represented the completion of medical instruction and the satisfying of tests of knowledge in the earliest days of our medical schools was that of bachelor of medicine, as it is today in some other countries. There is *a priori*, no reason why a bachelor's degree should not mark fittingly the termination of undergraduate study in medicine, as it does even now in the other learned professions of law and theology. However, in 1771, six years after its foundation, the University of Pennsylvania returned for the degree of doctor of medicine four men who had been graduated as bachelors of medicine in 1768. Reference to the catalogue of graduates of Harvard University shows that the bachelor's degree only was granted from 1788 until 1810, inclusive. With the granting of the doctorate of medicine, which now became the general practice, a higher degree in medicine became no longer possible. The degree of doctor of science has been bestowed, in recent years, upon doctors of medicine who have achieved eminence, although the degree itself is not distinctive. However, medicine is a branch of physical science, and something more, even if Bacon characterized it as the conjectural. But Bacon died in 1626, so, presumably, he should be pardoned for his unfortunate designation. So it has come about that distinction has been sought in degrees properly pertaining to the other learned professions, notably the degrees of doctor of laws and doctor of civil law, although so far as I know the degree of doctor of divinity or doctor of sacred theology has not been granted for distinction in medicine *per se*. Fellowship in the American College of Physicians has been safeguarded, so far as human foresight can go, and it is intended to be reserved for doctors of medicine who have achieved eminence in the field of internal medicine as practitioners and consultants, as investigators and scientists, and as authors and teachers. It is intended that fellowship in the American College of Physicians shall mean that its possessor has attained eminence in, and is an authority upon, internal medicine as a whole, or upon some of its recognized subdivisions. To define our meaning: "No one has reached a position of conceded eminence in his profession unless it is made to appear that he is deeply and broadly

educated, that he has made some substantial contribution to the literature of the medical profession, and that he has been entirely related to some phase of medical practice for a sufficient time to cause him to be widely recognized by intelligent members of the medical profession, as well as by a considerable number of people who have occasion to be interested in the services which that profession renders the people." The phase which concerns the college is internal medicine. "Authority in the medical profession is not acquired through a medical education that is only ordinary and a practice that is merely usual; eminence in the profession can be acquired only through the assiduous prosecution of medical practice for a considerable time, and through some special work, that has laid the profession under some obligation to the practitioner." Eminence and authority, as used in this connection, must be given a substantial and significant meaning.

The schools of medicine by their own action created an aristocracy in medicine as distinguished from law and theology; it has now become necessary for the internists to select from among their own number those whom they deem deserving of additional recognition. If this, with less reason, has been found necessary for the surgeons, of by no means distinguished scientific heredity, how much the more imperative is it for the internists that we shall recognize in a substantial manner those who have accomplished much in the upbuilding and the imparting of knowledge in the field of internal medicine!

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#### GRAHAM STEELL MURMUR

By J. EPSTEIN

New York

The heart and the lungs are most intimately associated both anatomically and physiologically through the pulmonary artery and the pulmonary valve, which form the connecting link in the cardio-pulmonary circulation. The structure of the pulmonary artery and its valve is of a more delicate texture than the aorta and is less liable to withstand increased blood pressure. The blood pressure within the pulmonary circulation is subject to various changes depending on the condition of both the heart and the lungs. Anything that will obstruct the free flow of blood within the pul-

monary circuit will cause an intrapulmonary hypertension and a rise of blood pressure in the pulmonary artery and in the right ventricle. If this condition be long continued it will lead to hypertrophy and dilatation of the right ventricle, with the consequent dilatation and stretching of the pulmonary ring and inability of the valve to close up completely the orifice during cardiac diastole. This imperfect closure of the pulmonary valve will cause a stream of blood to flow back from the lungs into the right ventricle during cardiac diastole and give rise to a diastolic murmur which is known as the Graham Steell murmur or a murmur of a relative pulmonary insufficiency. It is a soft blowing diastolic murmur heard best in the pulmonary area and in the third left intercostal space near the border of the sternum and is thence transmitted down the sternum. The second pulmonic sound is weakened or obliterated while the first sound may be accentuated. The murmur may vary in intensity or it may appear and disappear, according to the pressure variations in the pulmonary artery.

The common causes of obstruction in the pulmonary circulation which will give rise to an abnormally high intrapulmonary blood pressure are either diseases of the lung tissue, as emphysema, chronic interstitial pneumonia, pulmonary induration and general pleuritic adhesions, or diseases of the mitral valve, especially mitral stenosis, which causes back pressure in the left auricle and an excessive accumulation of blood in the pulmonary circulation.

Though a Graham Steell murmur is an uncommon condition, it is of considerable importance in the diagnosis and prognosis of some of the diseases of the heart and the lungs. The presence of a Graham Steell murmur, with indefinite physical signs of mitral disease, will point to a diagnosis of mitral stenosis, because it is the most frequent primary lesion in association with this murmur, and in every case of mitral stenosis the presence of a Graham Steell murmur should be looked for. When the mitral valve is intact and the left side of the heart is in good condition, the presence of this murmur will aid in the diagnosis of some chronic pulmonary disease. Conversely, when there is a mitral stenosis or a chronic pulmonary induration with a diastolic murmur at the base of the heart and a right ventricular enlargement, it will be in favor of a relative pulmonary insufficiency and not of an aortic insufficiency. A relative pulmonary insufficiency is of considerable prog-

nostic importance, because it indicates a great pulmonary engorgement which may ultimately lead to a tricuspid leakage and a general venous stasis with all the other evidence of cardiac failure.

An organic pulmonary insufficiency, in consequence of endocarditis and structural deformities of the pulmonary valve, though it is very rare, may follow such infectious diseases as acute rheumatic fever, pneumonia, scarlet fever and especially gonorrheal infection. The pulmonary valve may also become impaired through the extension of inflammation from an aortitis or an aortic aneurism. Stenosis of the pulmonary valve is mostly a congenital disease.

The differential diagnosis between a pulmonary insufficiency and an aortic insufficiency is not difficult. In a relative pulmonary insufficiency there is always present either a mitral stenosis or a chronic pulmonary fibrosis. It is associated with a right ventricular enlargement, a feeble pulmonary second sound, a clear aortic second sound and a small, quiet pulse. While in an aortic insufficiency, though the murmur may be heard with great intensity in the pulmonic area, there is the large left ventricle, the throbbing arteries, the Corrigan pulse, an obscure aortic and clear pulmonic second sound. To differentiate between a relative pulmonary insufficiency and an organic or valvular pulmonary insufficiency is rather difficult. A soft pulmonic diastolic murmur in the presence of a mitral stenosis or chronic pulmonary disease is in favor of a relative insufficiency, while a rough, harsh pulmonic diastolic murmur without any evidence of mitral stenosis or chronic pulmonary or pleuritic disease will favor a diagnosis of organic pulmonary insufficiency. In those rare cases where there is both an aortic and a pulmonic insufficiency, one may perhaps be able to discern two different diastolic murmurs, each in its respective place and of a different strength and quality. The aortic and the pulmonic second sound will be partly or wholly replaced by the murmurs and the heart will be enlarged on both sides.

Though a Graham Steell murmur indicates a disturbed cardiopulmonary circulation due to a lesion of the heart, or lungs, or both, which may ultimately lead to cardiac failure, a slight leakage of blood from the lungs into the right ventricle may serve a temporary useful purpose, tending to relieve an over-congested pulmonary circulation and prevent a general edema of the lungs.



BIOCHEMISTRY, IMMUNITY AND INFECTIOUS  
DISEASES

A CRITICAL REVIEW AND INTERPRETATION

By I. J. LEVY

New York

A DISCUSSION OF ACIDOSIS. With Special Reference to that Occurring in Diseases of Children.—J. Howland and W. McK. Marriott, Johns Hopkins Hosp. Bull., March, 1916.

The article gives an excellent résumé of the theory of acidosis and the methods employed to determine its presence. Authors point out that hyperpnea is the earliest clinical evidence of an acidosis and they lay particular stress upon the fact that an acetonuria may or may not accompany an acidosis. This fact has been generally recognized but has not been sufficiently emphasized by most men writing upon this subject. As they state an acidosis has been most thoroughly studied in diabetes, but it undoubtedly accompanies many other clinical manifestations. They were mostly interested in the acidosis accompanying recurrent vomiting and diarrheas in children and report remarkable therapeutic results by the simple administration of alkalis.

I was especially interested in the explanation of the acidosis accompanying the diarrhea of children, cases in which the abnormal acids (acetone and beta-oxybutyric) are absent in the urine and in which a striking symptom is a diminution in the output of urine. Since a high percentage of the acids accumulating in the blood are excreted by the urine this partial anuria is a rational explanation for a decreased alkalinity in the blood.

This explanation may suffice to account for the presence of an acidosis in some cases of true uremia, and suggests the reason why it is present only in the terminal stages. With a definite polyuria the acids are excreted in sufficient amount, but as the uremia proceeds, the resulting anuria is in all probability the cause of a beginning acidosis. If this fact is true alkali therapy can be of little use in uremia and this is borne out in most cases in which it is used. The fact that it is of such value in anuria accompanying a diarrhea of infancy is readily understood when we consider this type of anuria as being of extra-renal origin which it undoubtedly is.

RENAL DIABETES. D. S. Lewis and H. O. Mosenthal, Johns Hopkins Hosp. Bull., May, 1916.

In this article authors report a true case of "renal diabetes." They believe though the condition is rare there are a great number of cases that go unrecognized. They state the following two condi-

tions are necessary for a diagnosis. (1) A glycosuria, maintained at a fairly constant level and not markedly affected by changes in the carbohydrate content of the food. (2) A normal percentage of the blood sugar while the urine contains glucose. And they suggest that "renal glycosuria" is a more appropriate name.

The condition is due to an abnormal permeability of the kidney to sugar though in the case reported the patient presented no clinical evidence of nephritis and the various efficiency tests were normal with the exception of a slightly lowered phthalein output. It would be interesting to know if these cases presented an abnormal permeability to water and nitrogen, a condition which often is seen in certain types of glomerulo-nephritis and which would further substantiate the contention that these cases are a distinct type of nephritis.

Reports of such cases certainly emphasize the importance of estimating the percentage of sugar in the blood in all individuals excreting sugar in the urine.

It should also be borne in mind that a nephritic individual may only excrete sugar when the threshold point in the blood is considerably raised above normal. Many diabetics suffering with nephritis presenting no clinical manifestations, excrete a sugar-free urine though the percentage of sugar in the blood is far above the normal amount.

#### A COMPARISON OF THE ANTIGENIC PROPERTIES OF DIFFERENT STRAINS OF *BACILLUS TYPHOSUS*.—S. B. Hooker, *Jour. Immunology*, Dec., 1916.

Author has been able to demonstrate by cross complement-fixation tests that there are 3 distinct strains of the typhoid bacillus. By means of agglutinin-absorption experiments, he further corroborates his results and insists on the importance of employing a polyvalent typhoid vaccine when using such for prophylactic measures.

This grouping of the typhoid bacillus into 3 distinct strains throws some light upon the negative results obtained by many of us with the Widal reaction in clinical cases of typhoid. Most laboratories are employing a single strain in performing their agglutination tests, and are undoubtedly giving false serological reports on a great many typhoid infections.

The reviewer has had two interesting experiences this fall. In two cases of clinical typhoid fever in which the typhoid bacillus was isolated from the blood, the sera of the patients gave persistent negative Widal reactions with 10 different typhoid organisms (isolated from 10 different sources). Their own sera, however, strongly agglutinated the bacillus recovered from their blood.

I think with further study it will be shown that in various epi-

demics the percentage of positive Widal reactions will vary, depending upon the particular strain of typhoid bacillus which is predominant in any one epidemic.

THE PERSISTENCE OF ACTIVE LESIONS AND SPIROCHETES IN THE TISSUES OF CLINICALLY INACTIVE OF "CURED" SYPHILIS.—A. S. Warthin, *Am. Jour. Med. Sci.*, Oct., 1916.

In a study of 41 cases about one-third of a series of adult autopsies, author has demonstrated active syphilitic lesions in various organs, either by the presence of spirochetes or characteristic histological lesions. Even the cases which gave negative Wassermann reactions ante-mortem and were pronounced clinically cured gave evidence of syphilitic involvement in one or more organs. From this he concluded that a syphilitic is always a spirochete carrier and is a menace to the community.

That syphilis is a disease that is much more prevalent than was generally supposed no one who has had a close association with autopsy material could doubt. In a study of luetic aortitis for the past few years, I have been surprised at the number of individuals suffering with this condition, some giving clinical evidence of the trouble, others diagnosed only at the autopsy by histological examination of the aorta.

That Warthin has found spirochetes so successfully not only in organs with characteristic syphilitic changes, but also without any marked pathological changes is a fact of the greatest importance. However, one must be somewhat skeptical until this work is corroborated. The negative results encountered by other men both in Germany and the United States (Stadler, Gruber, Fukushi, Larkin, Levy) is not to be entirely disregarded.

Warthin, however, insists that he has spent in many instances a year before he could demonstrate a single group of spirochetes in some of the specimens. It may be true, therefore, that this exhaustive study has given him results which others could not obtain. Warthin states further that "the organs most frequently the seat of active lesions in order of frequency of occurrence would be the aorta, heart, testes, adrenals, pancreas, central nervous system, liver, and spleen."

The interesting feature of this is the frequency with which the aorta and testes are attacked, and the fact that the liver usually escapes. This should be borne in mind by the clinicians who are wont to consider every enlarged liver in an individual with a positive Wassermann as syphilitic.

## Progress of Diagnosis and Prognosis

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### GENERAL METHODS OF EXAMINATION—SYSTEMIC AFFECTIONS—DISORDERS OF GENERAL METABOLISM

**Determination of Sugar in the Blood**—G. L. WOLF and W. C. BALL, *Jour. Royal Army Med. Corps.*, Dec., 1916.

Authors describe a method for the determination of sugar in the blood which, though it has the disadvantage of being indirect and though it involves accurate measurements of both the cupric solution and the titanium trichlorid employed, it nevertheless gives a very accurate end-point determination. The titanium trichlorid used is a vigorous reducing agent and in so acting becomes oxidized to titanium tetrachlorid. The excess of trichlorid is then estimated with a known solution of ferric chlorid, using ammonium or potassium thiocyanate as an indicator. The method is given in detail in the original article.

SACHS.

**Blood Fibrin Formation in Diagnosis**—H. E. SMITH, *Med. Rec.*, Jan. 13, 1917.

Increased fibrin formation in the freshly drawn blood is found to accompany inflammatory conditions in which there is a production of a serofibrinous exudate. The technic is as follows: Place a small drop of blood on a very clean glass slide and carefully drop a very clean thin cover glass upon it. The cover glass should not be pressed down and the drop should be small enough so that when it spreads out it will not extend under the whole area of the cover glass. After five minutes, by means of an oil immersion lense, the red cells will be found to be grouped with open spaces between them. A specimen of normal blood will show only an occasional fine colorless strand of fibrin crossing the clear space. In blood with increased fibrin formation (hyperinosis), the strands of fibrin are more numerous, the increase being graded according to the strands from a small to a large increase. This test is of practical value in determining the presence of acute fibrinous involvement of the peritoneum in cases of acute appendicitis. One must exclude pneumonia, rheumatism, pus tubes, ectopic pregnancy, etc., any one of which would give the same fibrin picture as acute appendicitis. Hyperinosis in early pneumonia is a constant sign and it is also an indication of

peritoneal involvement in postoperative infections of originally clean abdominal cases. SACHS.

**The Angle of the Dropping Pipette and Accuracy in Agglutination Technique**—R. P. GARROW, *Lancet* (London), Nov. 18, 1916.

The number of drops and therefore the size of each drop, which a dropping pipette will deliver from a given quantity of a liquid varies considerably with the angle at which the pipette is held. For an equal variation of angle—e. g., 10 per cent.—the dilution error tends to be greater the further the angle is from the verticle. The only correct position in which to hold a dropping pipette is the vertical position. It is the easiest to adopt and maintain accurately and it is the least liable to error from slight alteration of angle. The drop method, when used intelligently is an extremely accurate method of making a series of dilutions for the estimation of the relative agglutinating titre of blood serum. SACHS.

**Leukocyte Reaction after Internal Hemorrhages**—H. DOLD, *Berliner klin. Wochenschr.*, Nov. 27, 1916.

Experiments on rabbits and dogs confirmed the observations made on guinea-pigs that there occurs a leukocytosis after sterile internal hemorrhages produced experimentally. This leukocytosis may occur after relatively unimportant internal hemorrhages. It attains its highest point about 8 to 24 hours after the internal hemorrhage. After 4 days it has entirely disappeared. This leukocyte reaction depends principally upon a reaction-leukocytosis and not upon a deficit-leukocytosis. The reaction can possibly be worked out to be of use in clinical diagnosis. MILL.

**Blood Examinations in Aviators**—E. MEYER and R. SEYDERHELM, *Deutsche med. Wochenschr.*, Oct. 12, 1916.

Aviators in service for over one year showed an increase in hemoglobin and erythrocytes. In some instances nucleated red cells were found. Inspissation was not observed. The aviators showed an increased generation of blood. MILL.

**The Urine in War Participants**—C. S. ENGEL, *Deutsche med. Wochenschr.*, Nov. 23, 1916.

Examination of 3210 urines. Of these 13 per cent. contained albumin. The albumin contents was mostly not large. There was a tendency that the albumin was increased in the summer. The diazo reaction was frequently positive before the universally introduced typhoid inoculation. Afterwards it almost suddenly ceased to be positive. Blood cells were found in numerous instances. MILL.

**The Pituitary Gland as a Regulator of Diuresis and the Specific Gravity of the Urine**—H. BAB, *Münchener med. Wochenschr.*, Nov. 28, Dec. 5 and Dec. 12, 1916.

A comprehensive article which does not lend itself to abstraction. Author concludes that the hormones of the endocrine glands influence the blood supply of the kidney and hence the diuresis. The pituitary gland exerts regulatory activity of the physiological functions of the urogenital system. Pathological polyuria is the result of a hyposcretion of the pars intermedia or of a disturbance in the posterior lobe of the gland. In diabetes insipidus the kidney appears to be healthy. A proof that renal concentration ability is not suspended is found in the fact that injections of the extract of the posterior lobe of the gland cause its normal activity. Hyperpituitarism can also not be accepted as an etiological factor in diabetes insipidus.

MILL.

**Composition and Physiologic Activity of the Pineal Gland**—F. FENGER, *Jour. A. M. A.*, Dec. 16, 1916.

The pineal glands of infants contain less phosphorus and more total nitrogen than adult glands. Pineal glands from cattle, sheep and lambs show only slight contracting power on unstriated muscle. This is very much less than that produced by equal amounts of the posterior lobe of the pituitary body, and not sufficient to be of physiological significance. Since the actions of the pineal gland, both on the blood pressure, the pulse rate and the excised heart, as well as on the uterine and intestinal muscle, are insignificant in therapeutic doses, and since health is not influenced by extirpation of the gland, it becomes difficult, at least with our present knowledge of physiologic chemistry to accept or even to consider the pineal body as an internal secretory organ of medicinal value.

SACHS.

**Studies in Creatin Metabolism**—F. P. UNDERHILL, *Jour. Biolog. Chemistry*, Oct., 1916.

Author concludes that creatinuria is not necessarily related to carbohydrate deficiency in the body. It is apparently associated with a condition of acidosis which is commonly concomitant with a metabolic state in which there is a carbohydrate deficiency.

SACHS.

**Three Cases of Acute Myeloid Leukemia**—A. ROSS, *Lancet* (London), Dec. 2, 1916.

In all of the 3 cases of acute myeloid leukemia, no very definite history of onset could be obtained, and it was usually the discovery of glands or of an enlarged liver and spleen that led to a blood examination and thence to a diagnosis. In 2 cases the mouth, teeth and gums were in a bad state, in the remaining one they were good.

In two cases very definite subcutaneous and other hemorrhages were clinically present, in the remaining case only a suspicious spot or two was observed. In all, the enlargement of the liver and spleen was only of a moderate degree. All had one or more sets of glands palpable and enlarged to a greater or lesser extent. In all the deficiency of red cells was great. SACHS.

**Fatal Case of Diabetes Mellitus Associated with Large-Cell Hyperplasia—**

J. R. WILLIAMS and M. DRESBACH, *Am. Jour. Med. Sci.*, Jan., 1917.

Authors draw the following summary: A case of fatal diabetes with large-cell hyperplasia of the spleen, lymph nodes, and liver is presented. It is the fourth case on record so far as authors can tell. Other cases have doubtless occurred, but the hyperplasia has been overlooked. It is highly desirable, therefore, that thorough autopsies be made whenever possible. The spleen, lymph nodes, adrenals, liver, and bone marrow especially should be examined, microscopically and with the polariscope, to determine the nature of the cell contents. The hyperplasia in the case described, and in the other cases reported, has some resemblance to the cell changes in Gaucher's disease of the spleen. In this disease lipoids are absent in the large cells, which, furthermore, have characteristic histological features. Moreover, Gaucher's disease is a familial disorder. It is shown that overfeeding with fat increases acidosis in diabetes and probably bears some relation, as yet not understood, to lipoidemia. SACHS.

**Diagnosis and Clinical Characteristics of Gout—**J. H. PRATT, *Boston Med. and Surg. Jour.*, Dec. 28, 1916.

Colchicum produces such a prompt relief from pain in cases of gout that it may be used as a diagnostic aid. Its action is not near so marked in acute rheumatism or in other conditions, which are likely to be confused with gout. The salicylates rarely produce any marked effect in controlling the pain of acute gout. While atophan produces a striking relief from the pain of acute gout, it also affords considerable relief in non-gouty arthritis. SACHS.

**The Teeth in Rickets—**J. L. DICK, *Brit. Jour. Child. Dis.*, Nov., 1916.

In rickets caries is exceedingly common, and hypoplasia, or defective calcification of the enamel is well marked. A hypoplastic condition of the teeth is characterized to the naked eye by a defective formation of the enamel and frequently stunted growth of the teeth. There may be only a pitting producing a honeycombed appearance of the enamel, or the enamel covering is slight and the cutting edge of the teeth presents sharp points, giving a characteristic appearance to the tooth. SACHS.

**The Cancer Problem**—G. W. CRILE, *Miss. Valley Med. Jour.*, Jan., 1917.

The diagnosis of cancer of the external parts is a comparatively simple problem. On the other hand the diagnosis of cancer of any of the internal organs is a complex problem. The clinical history of the patient, physical examination, the X-ray, blood tests, etc., may lead to a positive diagnosis of carcinoma, and the examination of the specimen removed at operation may prove the tumor to be benign; or a growth diagnosed as benign may prove to be malignant. It is unquestionable that the best diagnostic aid in cases beyond the reach of visual or manual examination is the X-ray. The Abderhalden reaction and hemolysis are of definite value in so far as they confirm clinical observations, but cannot be relied upon for final diagnosis, although it would seem that either or both may prove to be the stepping stones to simpler and more valuable methods for the diagnosis of cancer. As for the clinical symptoms—a loss of weight, gradual and slow at first, but steadily increasing, strongly suggests cancer. Any change in the size or consistency of an ulcer or a benign tumor is a sinister omen, especially when these changes are associated with loss of weight. In short, diagnosis depends upon the existence of definite changes in the form and function of the involved organ, upon changes in metabolism, and upon the alteration of any pathologic discharges. All the vast researches into the cause and cure of cancer have thus far yielded neither an adequate explanation of its primary cause, nor a specific cure. Until that specific cure is discovered, therefore, the one and only method of prevention is the removal of the pre-cancerous condition. The layman, laying aside morbid fears, must report to his physician any persistent abnormal visible growth or symptom of abnormal interest; and the internist must instruct his patient that the early and complete removal of the affected parts offers the only hope of cure, and with the surgeon, must carry on in the clinic and the laboratory researches for more definite methods of diagnosis and more efficient therapeutic agents. The surgeon, for his part, must develop his technic so that the cancerous growth may be completely removed with the least possible trauma, and must discover further means by which to conserve and increase the vital forces of his patients. As the cancer problem stands today, the disease is to be conquered only by the closest "team play" of these three—the layman, the internist, the surgeon.

SACHS.

## INFECTIOUS DISEASES

**Importance of Chronic Influenza in Surgery**—ARMBRUSTER, *Zentralblatt f. Chirurgie*, 1916, No. 45.

Chronic influenza with the typical, mostly only posterior, infiltra-



tions of the lungs plays an important part in surgery. It furnishes a good medium for streptococci; it favors the production of subcutaneous panarites or paronychites; it may give rise to erysipelas of rose-red coloration, and may cause inflammatory flat feet. Frequently it is associated with a pharyngitis and a furred tongue, and calls forth abscesses of the lymphatic glands. The healing process after operations is often interfered with and scars are liable to break. Of import are also the lumbar neuralgias after chronic influenza, as they may cause irritation of the cecum and even invagination.

MILL.

**Dysentery**—W. MAGNER, *Lancet* (London), Oct. 21, 1916.

A secondary invasion of the intestinal wall by bacteria from the intestinal lumen is an important factor in aggravating both the local and general condition in most cases of amebic and bacillary dysentery. This secondary invasion is the cause of the pyrexia observed in the later stages of amebic dysentery. Amebic dysentery may be latent, the ulcers being confined to the cecum and producing no symptoms. The earliest pathological change in bacillary dysentery is a dilatation of the blood vessels of the intestinal wall and a marked hemorrhagic exudation into the submucosa. Mannite-fermenting dysentery bacilli may exist in the intestinal wall in an avirulent form. The agglutination reaction in dysentery is a valuable means of differentiating the bacillary types of the disease.

SACHS.

**Spastic Constipation in Dysentery**—L. BORCHARDT, *Deutsche med. Wochenschr.*, Nov. 16, 1916.

The painful attacks in the lower abdomen which are characteristic of dysentery are due to spastic contractions of the colon.

MILL.

**Colitis Ulcerosa or Suppurativa**—R. EHLMANN, *Berliner klin. Wochenschr.*, Nov. 24, 1916.

The clinical picture of ulcerative or suppurative colitis, viewed in the light of modern laboratory and clinical experience, does not exist. In most of the cases formerly considered ulcerative or suppurative colitis we have really to deal with a chronic atoxic dysentery or at least with an ordinary instance of that disease.

MILL.

**The Rheumatic Child**—J. EPSTEIN, *Med. Rec.*, Jan. 20, 1917.

The symptoms and signs of rheumatism in a child may be atypical and indefinite. There may be an insidious onset with low fever and no sour sweats, no swollen joints, no severe pain, and no cardiac murmurs. But there may be a stiff neck, a sore throat, a slight muscle pain, or a dull aching joint, an irritable disposition, or a

slight incoordination of motion, a persistently raised or irregular pulse as the only evidence of rheumatism. SACHS.

**Acute Articular Rheumatism and Meningitis**—HERZOG, Deutsche med. Wochenschr., Nov. 2, 1916.

Case of a soldier in whom the meningococcus gave rise to a polyarthrititis as well as a meningitis. Recovery after intradural injection of serum. MILL.

**Complement Fixation Test for Gonorrhea**—T. P. SHUPE, Cleveland Med. Jour., Oct., 1916.

In this series of cases no case has been found in which the result was positive when the patient had not been infected with the gonococcus or did not have gonococci vaccine. Fresh sera must always be used, as serious errors on the positive side may come from a contaminated serum. A positive or negative Wassermann has no effect on the reaction. In acute cases when it is very easy to demonstrate the presence of gonococci, the fixation test is generally negative in the first 5 weeks and becomes positive from the fifth to the sixth week in uncomplicated cases. If the infection remains limited to the anterior urethra and subsides by the 7th or 8th week, the patient may never have a positive reaction. A positive reaction occurring in a person clinically cured of gonorrhea indicates the presence of a gonococcus focus, and these persons are potentially capable of infecting others. The antibodies may remain for 5 or 6 weeks after all organisms have disappeared and a negative test following a positive one in a supposedly cured case is good evidence that that patient is cured. About 30 per cent. in this series were positive when supposed to be cured. The test is of particular value in gonorrheal arthritis. It is about 100 per cent. positive in this class. In acute epididymitis, after 5 weeks from the onset of the disease, it is 100 per cent. positive. Chronic prostatitis, seminal vesiculitis and posterior urethritis give 80 per cent. positive reaction. An uncomplicated stricture does not give a positive result. An individual may have two or more infections and in interpreting a positive test it should be borne in mind that gonorrhea is a very widespread disease. SACHS.

**The so-called "Gonorrheal Heel"**—C. MAPES, Am. Jour. Surg., Feb., 1917.

Author states the following fundamental academic propositions: (1) That the terms gonorrhea (i. e., a flow of semen), blennorrhea and blennorrhagia (i. e., a flow of mucus), urethritis (i. e., inflammation of the urethra), gonococcus (i. e., semen berry), as at present almost universally employed, are totally inexact, inexpressive and fallacious, and should be eliminated from medical nomenclature; (2) that likewise the word gonorrheal (i. e., belonging or relating

to gonorrhea), wherever and however used, is distinctly erroneous, hence the expressions gonorrheal heel, gonorrheal exostosis, gonorrheal arthritis, gonorrheal iritis, etc., etc., are ridiculous and absurd; (3) that "gonorrheal exostosis of the calcar pedis" literally interpreted would be equivalent to saying that the afflicted individual had a flow of semen from the os calcis producing an outgrowth of the osseous structure, and most assuredly the rankest fanatic in the world will agree that this would be an anatomic, physiologic and pathologic impossibility; (4) that it has never been definitely demonstrated that the Neisserian diplococci are capable of inciting hypernutrition with consequent exostosis or osseous outgrowth in any anatomic situation, although systemic invasion of these microorganisms is frankly admitted; (5) that even were it possible to isolate the Neisserian diplococci from osseous excrescences excised from the calcar pedis or elsewhere, the causal relationship would by no means be thereby established, since exostoses have been encountered with greater frequency in patients not the subjects of Neisserian disease; (6) that the occurrence of osteophytes or exostoses upon the os calcis or elsewhere during or following an attack of Neisserian urethrorrhea is absolutely no proof of a necessary existing relationship, and to attribute the former to the malevolent influence of the latter is merely an expression of ignorance upon part of the observer; (7) that it has not been shown that exostoses or osteophytes in situations other than the calcar pedis ever occurred from invasion of the Neisserian diplococci, nor has it been demonstrated that these microorganisms possess an elective or selective affinity for the os calcis, as inferentially contended by certain authors; (8) that it appears strange that examination of the literature reveals no recorded instance of exostosis upon the calcar pedis in a female where any definite relationship was claimed between the osseous lesion and Neisserian disease, although it is well known other tissues embraced within the female organism are not exempt from the ravages of the diplococci of Neisser, in some instances there being infinitely greater structural damage than in the male; (9) that even admitting the premise that: (a) "periostitis, (b) hypertrophic osteo-  
periostitis, and (c) osteomyelitis" may be due to invasion of the Neisserian diplococci, the production of exostoses or osteophytes (i. e., new osseous formation) still remains unexplained; the pertinent fact cannot be ignored that during and following the inflammatory lesions mentioned osteoheliosis rather than osteogenesis almost invariably ensues; (10) that, finally, "gonorrheal heel" is equally as inexpressive and unintelligible as hematogenous kidney, acute abdomen, chronic appendix, and a thousand other not dissimilar diagnostic and anatomico-pathologic fallacies.

SACHS.

**Poliomyelitis**—B. SACHS, N. Y. Med. Jour., Dec. 23, 1916.

During an epidemic, every man's guess in regard to the probability of a case being one of poliomyelitis has at least 75 per cent. in its favor. The increase of the lymph cells and of the globulin content of the cerebrospinal fluid will corroborate materially the suspected diagnosis. The furibund cases always show either an absolute diminution of all the deep reflexes, a marked loss of tonus in the muscles, a marked hypotonia or in cases in which the meningeal symptoms preponderate, a slight increase of the reflexes and a slight rigidity of the neck and spine; also a difference determined by careful examinations, in the paralysis of certain groups of muscles as compared with others. SACHS.

**Communicability of Poliomyelitis**—C. G. KERLEY, Arch. Pediat., Jan., 1917.

Poliomyelitis may be communicated through personal contact. There are innocent carriers who spread poliomyelitis. Only a small percentage of children are susceptible. Probably from 90-95 per cent. of children possess an immunity. SACHS.

**Clinical and Serological Diagnosis of Typhus Fever**—A. SOUCEK, Feldärztliche Beilage z. Münchener med. Wochenschr., Dec. 19, 1916.

It is often very difficult to make a proper diagnosis of typhus fever on account of the great variability of the disease manifestations. The serological demonstration of typhus fever by means of the Weil-Felix reaction with x 19 is simple and reliable. MILL.

**Wassermann Reaction in Typhus Fever**—BITTORF, Feldärztliche Beilage z. Münchener med. Wochenschr., Nov. 14, 1916.

Author found a strongly positive Wassermann reaction in a case of typhus fever. Patient had no lues. When convalescent (6 resp. 7½ weeks later) the reaction was twice entirely negative. The same observation was made in another case. There was no evidence that either patient had had syphilis at any time. MILL.

**Diagnostic Value of Agglutinin Determinations in Inoculated Individuals**—E. W. A. WALKER, Lancet (London), Nov. 25, 1916.

In non-inoculated persons who have never had enteric fever (typhoid and paratyphoid infection) agglutination in a dilution of 1 in 25 justifies a strong suspicion of active infection. Inoculated persons, if quite recently inoculated, will usually show a high titre of specific agglutination. When an inoculated individual suffers from an attack of enteric fever, his titre of agglutination for the bacillus concerned (typhosus, paratyphosus A, or paratyphosus B) will exhibit the usual rise and subsequent regular fall seen in non-

inoculated subjects, but starting from and returning towards the higher base line of inoculated persons. The maximum of the curve of specific agglutinin formation accompanying enteric infection in man occurs between the sixteenth and the twenty-fourth day of the disease, and most frequently from the eighteenth to the twentieth day. In a case of mixed infections with enteric group bacilli (typhoid and paratyphoid), whether in inoculated or non-inoculated persons, the agglutinin curves for the different infecting organisms pursue their course independently of each other. SACHS.

**Typhoid Conjunctivitis**—L. JANKOVICH, *Deutsche med. Wochenschr.*, Nov. 23, 1916.

On the occasion of a therapeutic inoculation with non-carbolized typhoid serum, the acting physician accidentally received some of it in one of his eyes. A slight conjunctivitis developed from the secretion of which pure typhoid cultures could be grown after 24 hours. Another culture-test after 4 days was negative. However, after an incubation period of 3 weeks, the patient had a light attack of typical typhoid fever. MILL.

**Aural Typhoid Carriers**—A. B. BENNETT, *Jour. A. M. A.*, Jan. 6, 1917.

Author reports 2 cases of otitis media with a purulent discharge, containing living typhoid organisms. One case had a severe attack of typhoid fever 18 years previously and the other was the case of a child of 15 years of age who had never had an attack of typhoid fever. SACHS.

**Typhoid Fever and Pneumonia**—A. DÖBLIN, *Berliner klin. Wochenschr.*, Oct. 23, 1916.

While typhoid fever does not seem to tend to localize in the chest during peace, the association of typhoid with pneumonia appears to occur more frequently during war. Author reports 2 such typhoid-pneumonias which occurred sporadically, showing clearly the characteristics of the entire clinical picture. MILL.

**Isolation of Typhoid-Paratyphoid Bacilli**—A. LEITCH, *Brit. Med. Jour.*, Sept. 2, 1916.

In order to isolate the typhoid-paratyphoid organisms from the urine, author incubated for 24 hours equal quantities of urine and a solution of one part of brilliant green in 100,000 parts of peptone water. A loopful of the incubated material is spread on a Mac-Conkey plate and the organisms allowed to develop. Leitch treated 1,100 specimens of urine in this manner and on only one plate was an organism of the group isolated—a paratyphoid A. This method is a slight modification of the Browning-Mackie method. SACHS.

**Blood Picture in Pulmonary Tuberculosis**—K. TURBAN, *Zeitschr. f. Tuberkulose*, Vol. XXVI, No. 4.

The leukocyte picture is very important in pulmonary tuberculosis. It should be studied closely and recorded systematically. For the latter purpose author has devised a regular form and scheme. FRY.

**Recognition of Tuberculosis in Childhood**—J. HOLÓŠ, *Zeitschr. f. Tuberkulose*, Vol. XXVI, No. 5.

The prompt and definite recognition of tuberculosis in childhood facilitates its ready cure. The early recognition of the presence of tuberculosis is therefore the sine qua non of all tuberculosis treatment. It is essential that one observes: developmental disturbances, functional disorders of the alimentary organs, disturbances of the nervous system, headaches, vertigo, cardiac palpitation and other vasomotor manifestations, as epistaxis, inclination to perspiration and colds, and pains, especially in the cecal region. The manifestations on the part of puberty are usually at variance with those of the normal adolescent. FRY.

**Deposits of Medicaments in Tuberculous Structures**—STERN, *Zeitschr. f. Tuberkulose*, Vol. XXVI, No. 4.

The deposition of a medicinal agent in tuberculous tissues is dependent upon the distribution of blood within the structures. This deposition takes place when the blood current is slow and retarded, especially when the return circulation is interfered with. This mechanical process tends to explain certain facts which hitherto were considered to be of a chemotherapeutic nature. FRY.

**Fallacies in Diagnosis about Tuberculosis**—M. FISHBERG, *N. Y. Med. Jour.*, Dec. 2, 1916.

There is one fallacy which is responsible for a considerable part of the distrust of the laity for the medical profession. Author refers to the belief that if tuberculosis was invariably detected in its incipient stage, all patients would be cured. Most patients in the advanced stages of the disease blame their first doctor for all their troubles, and substantiate their grievances by the literature published by the societies for the prevention of tuberculosis in which it is distinctly and unequivocally stated that to cure the disease, it must be detected in its incipency, nay, in the so-called "pretuberculous stage," which can be done by the proper physician in all cases. It is a clinical fact, however, that these propositions are not altogether true. Author does not know of a single physician who, after applying all the scientific and empirical tests at the command of medical men, can tell who is likely to acquire tuberculous disease, on the

one hand, and who has sufficient resisting power to carry him through life without this malady on the other. He says this, knowing well that medical men are apt to tell some patients that they are "strongly predisposed to lung disease," and that unless vigorous measures are taken in time, there may be good reasons for regrets. Moreover, it is impossible to detect the so-called "pretuberculous stage," and in many cases it is more than difficult to detect active disease in its incipient stage. If we adopt the methods of diagnosis pursued by admitting physicians in sanatoriums, and consider every person who coughs, loses flesh, etc., and shows no other cause for such symptoms, suffering from a case of incipient tuberculosis, we shall detect thousands suitable for treatment; and we should fill sanatoriums with even larger numbers of patients who do not belong there. We must remember, however, that a diagnosis of tuberculosis should be made only when there are uncontrovertible proofs of the existence of active disease, and it is not always possible to discover these during the first visit of the patient. Indeed, in many cases it is necessary to keep the patient under observation for weeks before a diagnosis is arrived at. But even if we could diagnose all cases in their incipency, i.e., as soon as the patient begins ailing, we should not save many more than we do at present. Many cases, called acute, are advanced as soon as treatment is begun, and, no matter what is done for them, they perish within a short time. Others are subacute, and all treatment in vogue at present does not help them—they pursue a more or less rapid course and end fatally sooner or later; even sanatoriums, specific, dietetic, and climatic treatment fail to cure them. A very large proportion is of the chronic type. The patients last for years, and suffer several acute or subacute exacerbations, interspersed with several periods of "recovery." They are admitted to sanatoriums several times, each time as presenting incipient or moderately advanced cases, and no sooner are they discharged, as cured or with arrested disease, than they suffer relapse and have to be readmitted. That is not promptitude of recognition and the application of treatment which are always responsible for the cure of pulmonary tuberculosis, is clear when we bear in mind that autopsies show conclusively that more people recover from tuberculosis without treatment than suffer and perish from the disease. The belief that promptness in recognition and treatment of tuberculosis will eradicate the disease, is fallacious, and the sooner we realize this fact, the better for the patients and the profession.

SACHS.

**Relief Staining for Bacteria and Spirochetes**—T. H. C. BENIANS, Brit. Med. Jour., Nov. 25, 1916.

The advantages which author claims for his method of relief

staining are that the stain can be freshly made up with bacterium-free water and that it gives a clean and even background, which has very little tendency to form cracks and fissures. A small drop of a 2 per cent. aqueous solution of Congo red is placed on a slide, and a very small quantity of the bacterial culture is rubbed into it with the platinum wire; the drop is then spread out into a tolerably thick film. The film is allowed to dry and the slide then washed over with a 1 per cent. solution of HCl. in absolute alcohol and after being dried in the air, it is ready for examination. SACHS.

**Spirochetal Content of Spinal Fluid**—U. J. WILE, *Am. Jour. Syphilis*, Jan., 1917.

The spinal fluid from cases of early syphilis, of tabes and of paresis, contains spirochetes, as demonstrated by transplantation into the rabbit testis. The spirochetes may be present in moderate or even large numbers in the rabbit testis without producing the classic gumma or chancre of the testis. In some cases slight enlargement of the testis itself may be noticed. In still others spirochetes were demonstrated in which no increase in size of the testis was noted. In no case in this series were spirochetes demonstrable in the fluid itself before inoculation. The spinal fluid, at least in cases in which the nervous system is involved, must be regarded as infectious, and as such should be handled with the same care as other syphilitic secretia. SACHS.

**Surface-Tension and the Wassermann Reaction**—V. B. NESFIELD, *Lancet* (London), Jan. 6, 1917.

A simple definition of surface-tension is the force which drives a fluid against gravity up a fine capillary tube. Surface-tension is the all-important factor in the Wassermann reaction. Alcohol, bile, carbolic acid, and solutions of cholesterin, which act as antigens in the Wassermann test, all have very low surface-tension and greatly reduce the surface-tension of serum. It is difficult to distinguish a syphilitic antigen from a substance which lowers surface-tension. SACHS.

**Malignant Edema**—E. FRAENKEL, *Deutsche med. Wochenschr.*, Nov. 16, 1916.

The causative factors of malignant edema in man are of varying nature. Though they are all anaerobic, they differ from each other on account of their behavior when stained by Gram and on account of a number of other factors. MILL.

## RESPIRATORY AND CIRCULATORY ORGANS

**Anaphylactic Action of Grain on Respiratory Tract**—J. A. TURNBULL, *Bost. Med. and Surg. Jour.*, Dec. 28, 1916.

Author cites several cases of anaphylactic respiratory conditions in



bakers diagnosed by means of the cutaneous reaction to certain grains. This anaphylactic reaction to the proteid of grain is manifested on different parts of the respiratory tract in various ways. The entrance of these foreign proteids is by the gastrointestinal and respiratory tracts. The cutaneous reactions of wheat, barley, oats, corn and rice are demonstrated by author. A linear incision, half a centimeter in length, is made in the skin without drawing blood and at the same time the proteid is carried into the incised area. While immediate hypermia may appear due to mechanical injury, this disappears and positive reactions appear in from 2 to 20 minutes, manifested by the appearance of a wheal and hyperemia; burning and itching sensations may also be present. A control is made on the other arm.

SACHS.

**The Pulmonary Fissures and Lobes in Infants**—J. C. GITTINGS and A. G. MITCHELL, *Am. Jour. Dis. Child.*, Dec., 1916.

The fissures of the lungs in infancy show practically the same relation to the bony framework of the chest as in adults. The origin, course and termination of the fissures vary greatly in different individuals. The variations apparently do not depend on any of the anatomic characteristics of the chest and cannot be predicted therefrom. The lower level of the lungs in infants and probably in young children does not extend quite so low as in adults. The sixth interspace in the midthoracic line and the seventh or possibly the eighth interspace in the line of the angle of the scapula (at rest) represents the lowest limits of absolute safety for thoracentesis in early life.

SACHS.

**Reflex Cough**—A. B. BENNETT, *Med. Rec.*, Jan. 27, 1917.

Author divides the causes of reflex cough and the distribution of the nerves involved into three classes as follows: (1) Cough due to a reflex through the pneumogastric itself and through the pneumogastric and sympathetic combined; (2) cough due to a reflex originating along the glossopharyngeal and communicating directly with the pneumogastric by the communicating branch between the petrous ganglion of the glossopharyngeal and the root ganglion of the pneumogastric; (3) cough due to a reflex originating in the trifacial or other cranial nerve and communicating with the pneumogastric by way of the sympathetic nerve. Coughs originating from the glossopharyngeal are the ones most frequently noted. These coughs come from such causes as impacted cerumen or foreign bodies in the external auditory canal, and cholesteotoma, adenoids, enlarged tonsils, pharyngitis and pharyngeal papilloma, leptothrix, and lingual tonsil.

SACHS.

**Influence of Atmospheric Pressure upon the Blood Pressure in Man—**  
J. HÖHN, Wiener klin. Rundschau, Nov. 19, 1916.

There exists a certain connection between atmospheric pressure and blood pressure in man. In persons with increased blood pressure (arteriosclerosis) the pressure will decline when the individual changes his domicile to the seashore or to an altitude of medium height. In case the blood pressure be low, a residence in low altitudes will increase it. A marked influence, frequently causing increase of blood pressure, must be ascribed to the winds. Pronounced changes of atmospheric pressure are injurious to patients with a damaged vascular system. Author reports the case of an elderly patient who usually exhibited an increase in dysuria, oppression and brain congestion when southern winds prevailed. As soon as rain and colder temperature ensued these undue manifestations disappeared immediately. When the weather was warm and sultry the blood pressure was invariably and considerably increased.

MILL.

**Cardiac Aneurisms—**P. G. WOOLLEY, Jour. Lab. and Clin. Med., Jan., 1917.

The series of cases reported by author has to do with the formation of cardiac aneurisms. Cardiac aneurisms and aortic aneurisms resemble one another in every respect so far as pathogenesis is concerned and differ only in the fact that they appear in different organs. One of the cases reported showed the beneficial result of thrombosis in a heart which otherwise might have developed an aneurism; one showed an early stage of aneurism formation near the apex; one showed an early stage of aneurism formation in an unusual situation, and also illustrated calcification of the fibrous tissue forming the wall of the sac; one showed a rupture of a chronic aneurism in spite of a few pericardial adhesions; and one showed a fully developed large chronic aneurism.

WESTERN.

**Endocarditis—**H. K. DETWEILER and W. L. ROBINSON, Jour. A. M. A., Dec. 2, 1916.

The streptococcus viridans which authors isolated from the blood in cases of infectious endocarditis, is of very low virulence, probably lower than any hitherto reported as being recovered from a similar source. These streptococci are capable of producing lesions in animals identical to those found in the patients from whose blood the organisms were obtained. The streptococcus viridans isolated from the mouth of normal individuals are similar to those isolated from the blood of patients suffering from chronic endocarditis and are equally capable of producing heart lesions in the rabbit.

SACHS.

**Acute Cardiac Failure**—J. HAY, *Liverpool Medico-Chirurg. Jour.*, Vol. xxxvi, Parts 1 and 2.

Acute cardiac failure is due in very many cases to the onset of auricular fibrillation in hearts already handicapped by disease. The onset of the cardiac failure is sometimes so sudden and the downward progress so rapid that oral medication may prove too slow to be of any service. Again at times the patient's stomach will not tolerate any member of the digitalis group. A vicious circle is set up which ends in the death of the patient. SACHS.

#### ALIMENTARY TRACT

**Six Cases of Esophagectasia**—H. B. SHAW and A. W. WOOD, *Lancet* (London), Dec. 2, 1916.

Esophagectasia is the term applied to a dilatation of the esophagus which involves the greater part of the tube and is not due to the existence of a pouch. This condition is not very rare. In some cases hypertrophy of the muscle fibers of the esophagus and of the cardiac end is the rule, in others an atrophy exists, and in some there is a hypertrophy of one, and an atrophy of the other of these two structures. Such cases have in the past been frequently referred to as example of cardiospasm, by which is meant that quite independent of any organic lesion, spasm occurs in the circular muscular fibers around the cardiac orifice of the stomach. The symptomatology is very variable. When food does not pass down the esophagus properly, it "sticks" behind the breast bone and may even cause pain in the epigastrium; this sticking of food has been noted frequently for a number of years. Relief is got by making the food come back, by tickling the fauces, vomiting or by coughing or taking a deep breath. The taking of a meal is followed by a feeling of difficulty of breathing, described by some patients as a feeling of stifling or of asthma. In some cases the taking of a meal provokes a reflex cough. X-ray examination will show the condition present. These cases are apt to be confused with dyspepsia.

SACHS.

**Gastric Residuum**—C. C. FOWLER and Z. ZENTMIRE, *Jour. A. M. A.*, Jan. 20, 1917.

No striking differences between the gastric residuum of normal women and of normal men have become evident during the course of this investigation. The accepted limit of 20 c.c. for the volume of the residuum of the empty stomach of normal women is too low. An average volume of 49.44 c.c. was obtained in 81 cases. Both colorless and bile-colored residuums may be found. The two may occur in the same individual on different days or during the

same aspiration. The colored residuums are found more frequently in higher acidities. This is explained by the greater frequency of regurgitation in higher acidities. Total and free acidity vary directly with each other, free acidity rarely being found with a total acidity of less than 10, and always being found with a total acidity of more than 14.

SACHS.

**Duodenal Ulcer**—J. NOWACZYNSKI, *Deutsche med. Wochenschr.*, Nov. 26, 1916.

Three patients with supposed duodenal ulcer were found at operation not to be suffering from this condition. In 2 of the cases other gastric changes were found, while in the remaining instance a scar due to an old duodenal ulcer was met with.

MILL.

**Symptoms of Congenital Transduodenal Bands**—J. HOMANS, *Bost. Med. and Surg. Jour.*, Nov. 9, 1916.

Author reports 11 cases in which definite bands passing from the gall-bladder and liver across the duodenum were unassociated with gastric or duodenal ulcer or gall-bladder disease. These transduodenal bands produce a great variety of symptoms. In general the symptoms resemble those of gastric or duodenal ulcer or gall-bladder disease. In no instance was there a history of hematemesis or was blood found in the stomach washings. The average duration of symptoms was 8-9 years, but in one case gastric disturbance was only of 4 weeks' duration. In those cases in which a röntgen examination was made, the gastric and duodenal motility, position and outline showed abnormalities, but one is unlikely to mistake the X-ray findings of bands for those of cholecystitis, duodenal or gastric ulcer.

SACHS.

**Röntgen Indications for Surgical Procedure in Postpyloric Ulcer**—L. G. COLE, *Interstate Med. Jour.*, Jan., 1917.

Serial röntgenography reveals the progression or retrogression of the pathology in postpyloric ulcers, and this method of examination, connected with the clinical progress of the patient, gives a definite indication for the choice of medical or surgical treatment. Postpyloric ulcers evidenced by an obliterated cap and pyloric or postpyloric stenosis and broken gastric compensation demand surgical intervention. Postpyloric ulcers evidenced by an obliterated or deformed cap, and with pyloric or postpyloric stenosis and failing gastric compensation, require surgical intervention. Postpyloric ulcers evidenced by a deformed or obliterated cap without stenosis, but with a deep crater and thick edges, require surgical intervention. Postpyloric ulcers evidenced by an obliterated or deformed cap and secondary gastric involvement require surgical intervention, with removal of the induration. Postpyloric ulcers evidenced by an ob-

literated or deformed cap and with compensating peristalsis are a borderline group, where the choice of procedure must be determined by the clinical progress and subsequent röntgen examination to determine the progression or retrogression of the lesion. Postpyloric ulcers evidenced by simple cap deformity and with or without slight stenosis, and with a normal or compensating peristalsis and without deep crater or secondary gastric involvement, are more completely relieved of their symptoms by medical treatment than by surgical intervention.

SACHS.

**Gerlach's Valve and the Frequency of Epityphlitis in Vegetarians—**

COLLEY, *Archiv f. klin. Chirurgie*, Vol. CVIII, No. 1.

Gerlach's valve does in reality not exist. It supervenes when the angle formed by the appendix and the cecum is acute. In 152 Turkish bodies author found a normal Gerlach valve not in a single case. In more than two-thirds of the bodies he found, however, distinct signs of an old epityphlitis. He concludes that a disposition to appendicitis cannot be due to abundant meat ingestion, as supposed in certain quarters, as the Turks are largely vegetarians. MILL.

**Differential Diagnosis between Pneumonia and Appendicitis—A. L. GOOD-**

MAN, *Pediatr.*, Oct., 1916.

A most common error is to make a diagnosis of pneumonia because the patient exhibits a temperature, rapid respiration, possibly some dulness, and a slight cough. One must never forget that with every rise in temperature in a child there is always an increase in the number of respirations. Furthermore, one must always consider the possibility of a thickened pleura, the result of some of the former inflammatory condition in the chest. A slight catarrh, either acute or chronic, may account for an existing cough without any pathological change in the lung structure itself. Without bronchial breathing being detected, the presence of a pneumonia can be demonstrated only if one can show distinct signs of a beginning consolidation with the X-ray, indicating that the area consolidated has not yet reached a larger bronchus, and giving the distinct bronchial breathing characteristic of pneumonia. Abdominal pain with pneumonia is not an infrequent concomitant symptom. The pneumococcus is an organism which has an elective predilection for attacking the serous membranes of the body, and it is quite probable that the pain which one so frequently meets with in pneumonia is due to the same organisms which may set up some inflammatory change in the peritoneum itself. It is essential in order to make a thorough examination in a child, that the patient should be placed upon a bed or table, without any clothing whatsoever. It is well before making any physical examination to allow the child to remain on its back for a number of

minutes, and to watch and note carefully the character of the respiration. We know that in males the abdominal type of breathing predominates, while in females the thoracic type is more common. With the patient exposed, one may also note any local hyperemia; whether the intercostal spaces are shrunk or obliterated; whether the abdomen is held rigid or if there is free and flaccid motion of the abdominal walls; whether the patient will lie with the lower extremities completely extended, or whether there is a tendency to draw up the thighs; whether the patient prefers to be on one side or the other, and any little details which the observant physician will interpret in favor of one disease or the other. Another important diagnostic aid is bi-manual palpation. This is accomplished by introducing the well greased index finger of the right hand into the rectum, using the left hand for counter pressure on the abdominal wall. It is surprising how much can be felt in this manner, due to the smallness of the pelvis in children. It is well before introducing the finger into the rectum, to note the local condition existing there, for often fissures of the same anus give rise to marked abdominal pains, due to reflex irritation from the pain caused by the passing stool over the raw surfaces caused by these fissures. WESTERN.

**Lymphosarcoma of the Sigmoid**—A. L. GOODMAN, Arch. Pediatr., Oct., 1916.

Report of a case in a child, 4 years old. The little patient complained of chronic constipation and bleeding from the rectum, accompanied with extreme pallor and marked weakness, which commenced about three weeks before admission. At the same time, that is, three weeks before admission, the mother noticed a hard lump in the baby's abdomen. The child was in very good health up to four months before its entrance into the hospital, when the mother noticed that the child had become somewhat constipated. Previous to this time the bowels had been fairly regular. About two weeks before the appearance of the blood, the mother noticed that the child would have severe abdominal cramps before an evacuation of the bowels. During the three weeks previous to her admission there would be severe straining at stool, but no fecal matter would be expelled, but at times small quantities of pure blood. At one time there was as much as half a pint of pure blood passed during one of these severe attacks of straining while the child sat on the vessel. These attacks became more frequent, and for the three days preceding her admission, the child would have two or three movements of pure blood during the day. The mother noticed that the lump in the abdomen would change its position from day to day, and that it had grown larger in size. Clinical Considerations—We have a child with a rather acute history of three weeks' duration, at which time it was seized with sudden abdominal pain, so severe

in type that the child lay upon the floor and screamed with pain. The mother picked the child up, noticed the abdominal tumor and shortly afterward the child passed blood from the rectum. The blood was dark in color and clotted. From our past experience we have been taught that dark clotted blood passed from the rectum, indicates that the blood comes from high up in the intestine. The sudden appearance of the tumor, its sausage-shaped character, would lead one to consider in the differential diagnosis, the possibility of an intussusception of the intestine. Against the intussusception, were the painlessness of the tumor, the absence of vomiting, and the duration of the swelling, as well as the general glandular enlargement which spoke more in favor of a new growth. The negative von Pirquet, no signs of a general tuberculosis, excluded the possibility of a tuberculous condition of the omentum. With a constant loss of blood, the increasing pallor, the gradual increase in size of the swelling with its accompanying glandular involvement, indicated operative interference. An X-ray examination revealed a distinct obstruction in the large intestine which apparently was in the distal portion of the transverse colon. The röntgenologist stated that from the character of the shadow he felt confident that the case was one of malignancy. On February 28th, the child was operated, and a three-inch midline incision was made over the region of the tumor. Upon exposing the abdominal contents, a tumor, about six inches in length, and three inches in width, of the sigmoid, was found with metastatic lymph nodes in the mesentery, also surrounding the gut. The pathological report confirmed the clinical findings.

WESTERN.

**Acquired Hemolytic Jaundice**—G. A. FRIEDMAN and E. KATZ, *Jour. A. M. A.*, Oct. 28, 1916.

The anemia in hemolytic icterus is not, as a rule, severe. In an analysis of 158 cases of both types of hemolytic jaundice in which blood counts were made, Krumbhaar found that 55 belonged to the acquired form and 103 to the congenital or familial type. The average red cell count of the 55 acquired cases was 2,032,200; the counts ranged from 1,000,000 to 4,500,000; 10 cases showed a count which was below 1,000,000. In 27 the count was below 2,000,000, and in only 4 cases was it over 4,000,000. Pearce and his co-workers have shown that after splenectomy in dogs a definite and prolonged anemia is produced. After splenectomy in hemolytic jaundice, however, the anemia disappears. It seems probable that the removal of a spleen that has undergone some pathologic change is followed by entirely different results from those which follow the removal of a normal spleen.—The chronic slight icterus so characteristic of the disease is the direct result of the destruction of the

blood. The manner in which this jaundice is produced has been thoroughly explained. It is due to the obstruction in the bile passages caused by the extreme viscosity of the bile. The sequence in which this occurs is briefly as follows: By whatever agents produced, the red blood cell is destroyed with the liberation of the hemoglobin. This is then split into two parts; first, the iron-containing portion, or hematin, which is carried to the liver (Kupffer cells), bone marrow, kidney and other organs; second, the pigment-containing portion, or hemochromogen, which is taken up and excreted by the liver as bilirubin. Thus there is an increased formation and excretion of the bile pigments with an increased viscosity of the bile, which causes an obstruction to the flow of the bile in the smaller ducts. Reabsorption of the bile takes place, leading to the production of jaundice. The bilirubin may be demonstrated in the blood serum. The bilirubin in the intestines is converted into urobilinogen by the reducing action of the intestinal bacteria. This is absorbed and excreted by the kidneys. Urobilin and its antecedent, urobilinogen, may be demonstrated in the stools, and as has been suggested by some in the duodenal contents. The amount of blood destruction has been estimated by the amount of urobilin and urobilinogen in the urine and in the stools. Eppinger assumed that one molecule of urobilin represented one molecule of destroyed hemoglobin. Addis, however, has described a method for determining accurately the quantity of urobilin and urobilinogen excreted in 24 hours. The splenomegaly is one of the principal characteristic features of the disease. The findings in the spleens, both in those removed by operation and in those which were obtained at necropsy, showed very little proliferation, if any, of the connective tissue. There was frequently great loss in the number of the follicles, and in a majority no germinal centers were to be seen. The splenic sinuses were dilated. Constant infiltration of the pulp with red blood cells was perhaps the most characteristic feature noticed. The endothelium of the blood vessels was proliferated and was filled with coarse hemosiderin. On necropsy nothing definite was found. The liver, however, showed a deposit of pigment, and this was also seen in the kidney and bone marrow. Eppinger showed the presence of an enormous hyperemia in the spleen sinuses of pernicious anemia, as well as in hemolytic icterus. Pribram has pointed out the fact that stasis of blood in the spleen, as caused by chronic passive congestion, is associated with an increased destruction of red blood cells. He also showed that urobilinuria could be easily caused by inducing congestion of the spleen. He therefore associated increased blood destruction, anemia and urobilinuria with stasis of the spleen.

SACHS.



**Chronic Obstructive Jaundice**—J. F. ERDMANN and C. G. HEYD, *Am. Jour. Med. Sci.*, Aug., 1916.

A neoplasm at the ampulla of Vater either by its presence, by kinking of the duct, or associated edema of the mucous membrane of the duodenum or common duct will bring about not only biliary obstruction but a variable degree of pancreatic obstruction. The degree of obstruction to pancreatic secretion will depend upon the individual anatomical topography of the ducts of the pancreas. In about 83 per cent. the duct of Wirsung carries the entire pancreatic secretion. In about 12 per cent., however, the duct of Santorini is the main duct, while in 54 per cent. the duct of Santorini may act as a substitute for the duct of Wirsung. In certain cases the duct of Santorini might remain uninvolved for a considerable period of time, and, moreover, the duct of Santorini is not infrequently connected with the duct of Wirsung, and thus it is possible for a drainage of the pancreatic secretion to take place into the duodenum even with almost complete biliary stasis; in fact, there may be complete biliary stasis with little or no pancreatic retention. Certainly, this must be an extremely rare condition, and, aside from its theoretical interest, does not possess any particular technical importance over the general run of cases. Cholecystenterostomy will deliver the biliary secretion or excretion into the intestinal tract. The stools would contain bile and would present an approach to normal coloration, and there would be a cessation of the jaundice and an absence of bile in the urine. This operation, however, could only rarely affect the pancreatic secretion, and the few cases that it did influence, pancreatic drainage would depend upon retrograde flow of pancreatic juice into the common duct, then through the cystic duct and gall-bladder into the intestinal tract. In the large majority of cases the patients suffer from a lack of pancreatic secretion in the intestine with a corresponding lack of pancreatic digestion, as evidenced by the bulky frequent stools, showing the increase in the total amount of fats and the changed relationship between unsaponified and saponified fats and the absence of adequate proteid digestion. Therefore, in spite of the elimination of jaundice and the biliary stasis by cholecystenterostomy, any or all of these cases may proceed to a more or less rapid death as the result of pancreatic inefficiency. Any chronic obstructive condition of the duodenum below the ampulla of Vater will introduce in addition to the signs of biliary stasis those of pyloric stenosis, and in two of the cases presented herewith the clinical picture was that of chronic pyloric stenosis and chronic obstructive jaundice. In one of author's cases there was a distinct interim between the syndrome of pyloric obstruction and a second syndrome of biliary stasis. In this particular case a malignant ulcer existed, perhaps primarily, of the duodenum below

the ampulla of Vater, and for which a posterior gastro-enterostomy had been performed. Subsequently, the ulcer more thoroughly invaded the ampulla and brought about biliary obstruction, for which a cholecystenterostomy was performed. In obstructive conditions at the ampulla of Vater it is usual to find the gall-bladder distended with bile. (Courvoisier.) This is not necessarily always the case, as a distinct hydrops and a well-dilated common duct filled with clear mucoid fluid have been observed, and when this rather uncommon condition is seen it is associated with patulous cystic and hepatic ducts and mechanically represents a pressure acholia. Kausch made a careful study of such a case: "At operation upon a patient with steadily increasing jaundice the gall-bladder and common duct were found enormously distended with clear fluid; a cholecystostomy was performed and for two hours clear fluid flowed from the tube. Then the discharge began to be slightly colored, and by the end of six hours it had assumed the appearance of somewhat pale bile, large quantities of which continued to flow as long as the sinus was left open. At autopsy, some weeks later, a small tumor was found at the papilla of Vater. Kausch thinks that the hydrops in these cases is due to excessive secretion by the mucosa of the gall-bladder and ducts, whereby the duodenal opening being occluded, the pressure in the biliary system being so raised that the bile secreted by the liver cells is poured, not into the excretory ducts, but back into the blood and lymph vessels of the liver. The most frequent obstructive condition is from carcinoma of the pancreas, ampulla, or duodenum. Pancreatic cancer is the most rapidly fatal of any form of carcinoma; death ensuing within 7 or 8 months from the time of onset of noticeable symptoms, and occurs usually before the growth metastasises or obtains any great local extension. SACHS.

**Actinomycosis of the Liver**—SIMMONDS, Aertzlicher Verein in Hamburg, Münchener med. Wochenschr., Dec. 19, 1916.

Actinomycosis of the liver may appear in tumor form without suppuration. The tumor is well circumscribed, tough, resembling a sarcoma. The character of the tumor is determined by its central portions which are soft, yellow and filled with granulated masses. Author showed preparations of 4 cases. In these cases the infection took probably place through the gastrointestinal tract. The prognosis is bad. The diagnosis cannot be made before laparotomy is performed, as fistulas do not occur, as a rule. Serologic methods may be of service in rendering an early diagnosis. MILL.

## NERVOUS SYSTEM

**Absence of Patellar Reflexes without Organic Disease of the Nervous System**—R. V. HÖSSLIN, Münchener med. Wochenschr., Nov. 14, 1916.

Observation of 2 cases in author's private institution. Case I

was a woman, 37 years old. Was emaciated and had pronounced cardiac palpitation. No luetic history. Wassermann negative. Spinal fluid could not be examined. Very careful examination of the nervous system gave only negative results; however, patellar as well as tendon Achilles reflexes were absent on both sides. Case II. A woman, 58 years old. Showed extra-systolic irregularities and other cardiac shortcomings. Very exact examination of nervous system could not elicit any organic disease of the same. Absence of patellar and tendon Achilles reflexes. No history of syphilis. Wassermann in the blood negative. Spinal fluid could not be examined. The last patient knew that she never had these reflexes and that none of her three brothers had ever had them. In three brothers of her father the patellar reflexes had also always been absent.

MILL.

**Gunshot Wounds of Peripheral Nerves**—B. STOOKEY, Surg. Gynec. and Obstet., Dec., 1916.

With the use of high explosives and bullets with high velocity, the frequency of nerve lesions has increased. The nerve may be injured primarily by direct violence or secondarily by scar tissue or callus. Diagnosis cannot be made before operation between anatomical and physiological division. Diagnosis can usually be made in cases with incomplete division. Musculospiral nerve injury in its lower third shows loss of sensation on a narrow band or over the dorsum of the thumb, usually only a loss to cotton wool and temperature sense. An injury to the musculospiral nerve may cause dissociation of temperature sense in the area on the dorsum of the hand without loss to cotton wool. Trophic ulcers occur only after trauma.

SACHS.

**Sensory Disturbances of Cerebral Origin**—A. GORDON, Arch. Int. Med., Dec. 15, 1916.

Superficial sensations are to a greater or lesser extent involved in all the lesions of the brain considered in this article. The capsular lesions give the most pronounced and the most persistent disturbances in the touch, pain and temperature senses. The least involvement is seen in the cortical cases. In spite of the apparent regularity in sensory impairment, certain exceptions nevertheless present themselves so that it is apparent that certain conservative reservations must always be made when diagnostic inferences are to be deduced.

SACHS.

**Spinal Fluid Findings in Cord Compression**—J. B. AYER and H. R. VIETS, Jour. A. M. A., Dec. 9, 1916.

Authors believe that changes in the spinal fluid frequently occur

as a result of compression of the spinal cord. The principal characteristic of such compression fluid is marked increase of proteins without corresponding cellular increase, obtained under normal pressure. Xanthochromia with massive coagulation added to the above fluid makes a more intense reaction of the same significance, only more conclusive.

SACHS.

**Multiple Spontaneous Intracerebral Hemorrhages, a Contribution to the Pathology of Apoplexy**—P. GREENACRE, Johns Hopkins Hosp. Bull., Feb., 1917.

The following conclusions are offered by author: In the examination of an adequate number of brains containing multiple spontaneous hemorrhages, pontine hemorrhage was found secondary to extensive hemorrhage into the internal capsule and basal ganglia in a majority of cases. The liability of the pontine arteries to secondary rupture is due probably to their anatomic peculiarities in that they are short, small, terminal branches given off nearly at right angles from a large trunk, and possibly to a disturbance of blood pressure in the circle of Willis with a back-flow into the posterior branches.

SACHS.

**Blood and Blood Vessels in Epilepsy**—T. E. SATTERTHWAITE, N. Y. M. Jour., Nov. 11, 1916.

Abnormalities of cardiovascular phenomena occur in the vast majority of epileptic seizures. The grosser forms of cardiac disease rarely occur in epilepsy. In fact, they are present in so small a proportion as to indicate that they are accidental rather than determining factors of it. That a cerebral disease or abnormality may produce epilepsy is well established. The evidence shows that removal of enlarged veins or nevoid growths adjacent to the base of the skull has been followed by cessation of the seizure. There is therefore a *reciprocal* relation between circulatory disorders and epilepsy to this extent; that epilepsy causes circulatory disturbances and that abnormalities of blood or vessels cause epilepsy. This reciprocal relation I believe to have been hitherto overlooked. In most forms of epilepsy there is cerebral anemia, and this is relieved effectively by various heart stimulants, the high frequency current, and radiant electric light. The importance of the use of cardiac stimulants in epilepsy, author believes, has not been properly appreciated by the profession.

SACHS.

**Early Diagnosis of Tabes Dorsalis**—W. F. SCHALLER, Jour. A. M. A., Jan. 20, 1917.

In tabes the syphilitic posterior leptomeningitis of the spinal cord

(and probably also of the radicular nerve) is in etiological relationship to the degeneration of the posterior columns of the cord. Every case of syphilitic posterior leptomeningitis and consequent multiple symmetrical radiculitis is a case of potential tabes. This meningeal reaction is evidenced in the cerebrospinal fluid by an increased cell count and increased globulin content; and probably by a positive Wassermann reaction indicating that this reaction is syphilitic in nature. It is necessary to make repeated lumbar punctures in doubtful cases. Syphilitic radiculitis has its characteristic disturbance of the sensibility: Subjective disturbances are pain and paresthesia; objective disturbances are deep and superficial sensibility impairment, the latter following radicular distribution. In a patient with a history or other evidence of syphilis presenting characteristic sensibility disturbances of the radicular type with a tendency to symmetry, one should suspect a potential or early tabes. If associated with the foregoing, we have a positive reaction in the cerebrospinal fluid indicating a chronic syphilitic meningitis together with such pupillary phenomena as anisocoria, pupillary irregularity or sluggish reaction to light, the diagnosis of early tabes is most common.

SACHS.

**Cerebellar Localization**—J. L. MEYERS, *Jour. A. M. A.*, Dec. 9, 1916.

The function of the cerebellum is to inhibit, control and regulate the activity of the motor cortex of the cerebrum and the paracerebellar nuclei in the medulla. The phenomena of cerebellar sufficiency are, accordingly, to be interpreted as phenomena of hyperfunctional and not hypofunctional activity. The cerebellum is primarily related through its various lobules to the various motor centers in the cerebrum, and the tonus centers in the medulla. SACHS.

**Cerebellar Abscess, Differential Diagnosis**—P. D. KERRISON, *Laryngoscope*, Dec., 1916.

Differential points between cerebellar abscess and temporo-sphenoidal abscess. Recurring vomiting, common in cerebellar abscess, is exceedingly rare in cerebral abscess. Nystagmus, a frequent phenomenon in cerebellar abscess, is never caused by cerebral abscess. Characteristic cerebellar ataxia with tendency to fall constantly in one direction is peculiar to that lesion, though some disturbance of equilibrium due to increased pressure may occur in cerebral abscess. Unilateral incoordination ataxia and diadokokinesis, not uncommon in cerebellar abscess, are not characteristic of cerebral abscess. Hemiparesis, occasionally present, is most often homolateral in cerebellar abscess, whereas in cerebral abscess, it is always contralateral. In cerebellar abscess both upper and lower

limbs are involved; in temporal lobe abscess, the leg usually escapes. Speech defects, drawl, slow "syllabic" speech, lisp, are occasional symptoms of cerebellar abscess, which are not characteristic of cerebral abscess. An unclouded mentality is characteristic of cerebellar abscess; mental dulness, almost to obscurity but without perversion, and frequently retarded or delayed cerebration—are characteristic phenomena in cerebral abscess. Various forms of aphasia, and occasional contralateral paralysis, which occur in some cases as focal symptoms of cerebral abscess, do not occur in cerebellar abscess. When focal symptoms are present, the above lesions may be easily distinguished; when absent, a positive differentiation may be difficult or impossible. Differential points between cerebellar abscess and acute diffuse suppurative labyrinthitis. Cerebellar abscess resembles labyrinthitis only when nystagmus and associated phenomena are present, and then only the acute stage of suppurative labyrinthitis. When the acute stage of suppurative labyrinthitis has passed, all similarity disappears. Nystagmus in diffuse suppurative labyrinthitis is always toward the sound ear; in cerebellar abscess, it may be alternating or in either direction, but is usually strongest toward the side of the lesion. Nystagmus tends to subside rapidly in labyrinthitis,—it is persistent and sometimes progressive in cerebellar abscess. Disturbances of static equilibrium: In acute suppurative labyrinthitis, the patient tends to fall always in the direction of slow nystagmic movement and therefore toward the diseased ear. In cerebellar abscess, as the disease advances, the falling direction is often quite independent of the nystagmus, and is constant in direction. There is absolute deafness of the involved ear in diffuse suppurative labyrinthitis; in cerebellar abscess the hearing is not impaired unless the abscess is secondary to a suppurative lesion of the labyrinth. Caloric test: Vestibular apparatus does not respond to irritation by heat or cold in suppurative labyrinthitis; it responds normally in cerebellar abscess uncomplicated by labyrinthine disease. All the characteristic symptoms of the acute stage of suppurative labyrinthitis tend to subside rapidly. The symptoms of cerebellar abscess are persistent and tend to increase rather than subside.

SACHS.

**Prognosis in Disseminated Sclerosis**—B. BRAMWELL, Edinburgh Med. Jour., Jan., 1917.

Author analyzed 200 cases of disseminated sclerosis and reports the results, as regards prognosis, as follows: 106 of the 200 patients have died; 64 patients are known to be still alive, and in 30 cases the result is not known. The average duration of the disease in 170 fatal and non-fatal cases, up to the present time, is 12 years and 1 month. In the 106 cases in which the disease proved fatal, the

average duration of life was 10 years and 8 months; the shortest duration was 7 months, and the longest duration was 37 years. Two cases are cited in whom a permanent cure seems to have taken place.

SACHS.

**Etiology of Congenital Mental Deficiency**—W. A. T. LIND, Med. Jour. Australia, Oct. 14, 1916.

Author investigated 317 cases of congenital mental deficiency. He states that such mental deficiency may either be caused by a phylogenetic degeneration or by disease or trauma of the brain in early life. Both of these types of mental deficiency may result in idiocy. From the investigation of these cases, author is undecided whether alcoholism is an indication of stock degeneration or one of the causes which produce phylogenetic degeneration.

SACHS.

**Psychology of Malingering and Functional Neuroses**—T. LUNSDEN, Lancet (London), Nov. 18, 1916.

Hysteria is to be regarded as a disorder of the subconscious phase of the personality, in which suppressed desires and emotions manifest themselves vicariously by symptoms referable to any or all of the cerebral activities, motor, sensory, vaso-motor, or sympathetic. In hysteria the highest conscious centers and cells lose their control over the lower cerebral centers, which then act under the direction of the subconscious and emotional areas. The motives being denied outlet in their normal direct course find expression in some perverted and abnormal direction. Neurasthenia is the state which results when the brain cells are deficient in "ergogen." Ergogen is a term applied to the balance of nerve energy upon which the state of well being in this direction depends. The patient is bankrupt as far as energy goes, yet hypersensitive of different impulses reaching the brain. Rest and nourishment restore the balance of ergogen and so cure the condition. Malingering connotes the fraudulent feigning of symptoms or signs of the disease selfishly for gain, and is a disease of the highest conscious cerebral centers in the frontal lobes.

SACHS.

**Symptomatic Psychoses**—E. S. BRODSKY, Med. Rec., Jan. 20, 1917.

To the symptomatic psychoses belong those mental disturbances which are, as Southard defines them, of somatic but "extranervous" origin. Infections and autointoxications are the etiological factors. Three cases are cited: (1) A case of delirium with prevalent maniacal symptoms due to a septic process complicated in the first attack with septic endocarditis; in the second attack the physical condition is characterized through marked leukocytosis, irregularities of heart action, elevation of temperature, absence of menses, and pain in

the arms, evidently the result of a toxic neuritis. (2) A case of delirium with complete disorientation for time and place and partially for person. This case was due to disturbed heart compensation. The gradual deepening and clearing up of consciousness indicates the toxic character. (3) This case was associated with malaria. The mental symptom-complex represents disorientation for time and partially for place and person. The patient recovered rapidly after quinine treatment. SACHS.

#### URINARY ORGANS—MALE GENITALIA

**Contracted Kidney**—F. HIRSCHFELD, *Berliner vereinigte ärztliche Gesellschaften, Münchener med. Wochenschr.*, Nov. 15, 1916.

Clinical observations concerning contracted kidney. On a diet containing 40 grams albumin and 5 grams sodium chlorid, besides the other food-essentials, the amount of urine declined. Rest in the sun caused a further decline in the amount of urine. During the day, when exposed to the sun, the specific gravities were highest. The kidney seems to regain its former elasticity under the influence of the warmth and a limited intake of proteid and salt. The albuminuria became also diminished. In other respects the influence of this treatment was also beneficial. The headaches became less severe and the bodily vigor and resistance increased. A diet poor in salt not only reduces the amount of urine but also increases perspiration. Although polyuria is a compensatory process, the observations of author show that the absence of this condition causes improvement of the renal activity. Chronic contracted kidney permits of the favorable diagnosis of renal sclerosis provided the irritative processes have been removed. MILL.

**Cystic Kidney**—W. CARO, *Deutsche med. Wochenschr.*, Oct. 26, 1916:

The case of a soldier, 37 years old. The clinical diagnosis was renal concretion with secondary pyonephrosis. The extirpated left kidney proved to be a congenital cystic kidney. As this malformation invariably appears bilaterally, the other kidney, which palpation showed to be also enlarged, could not be simply hypertrophic, but had to be considered to be also of a cystic nature. A grave uremia ensued after nephrectomy which rapidly disappeared after the administration of abundant sodium chlorid. Death ensued at the end of the fourth week as a result of hypostatic pneumonia. MILL.

**Tuberculosis of the Kidney and Menstruation**—W. HOFMANN, *Berliner klin. Wochenschr.*, Nov. 6, 1916.

Author describes the premenstrual type of the fever in renal tuberculosis and the critical decline of the fever when menstruation en-



sues. The onset of the menses is also generally followed by an improvement of the general condition. MILL.

**Experiences in Renal Surgery**—W. TAYLOR, Dublin Jour. Med. Sci., Dec. 1, 1916.

Where movable kidney is unquestionably the cause of the symptoms complained of by a patient or, when the mobility is leading to actual renal pathological changes, operation (nephropexy) is undoubtedly preferable to the use of any mechanical apparatus. The operation is devoid of risk. Calculi once diagnosticated should be removed by operation without delay, otherwise destruction of the kidney will inevitably occur sooner or later. The mortality following nephrotomy in aseptic cases is not more than one per cent., whereas the mortality of nephrectomy for calculous disease is about 17 or 18 per cent. The prognosis of nephrectomy for hypernephroma, if performed before secondary deposits have occurred in other parts of the body, is exceedingly favorable. SACHS.

**Anuria for 5½ Days in a Patient on whom Left Nephrectomy had been done**—G. J. THOMAS, Journal-Lancet, Dec. 1, 1915.

The points of interest in this case are the following: (1) Complete anuria for 5½ days, and the total amount of urine secreted in nine days 64 ounces. (2) The patient had but one kidney; the other having been removed some time before for pyomemphrosis. (3) No history of pain during the present attack. (4) The cause of obstruction not known. No stones or fragments were passed before or after cystoscopy. Röntgen ray was negative. (5) Relieved by permanent ureteral catheter. (6) The patient in apparently normal health after 10 days, and no damage to the kidney demonstrated. SACHS.

**Röntgenographic Diagnosis of Renal Calculus**—J. FRIEDMANN, Am. Jour. Röntgenology, Sept., 1916.

In reno- or uro-röntgenography special precautions must be used to avoid errors, as many extra-urinary bodies may be taken for urinary calculi. They are: (1), phleboliths in the pelvis; (2), calcified plates in the arterial wall; (3), calcified lymph nodes; (4), calcareous glands; (5), tuberculous glands in the mesentery; (6), calcified portions of costal cartilage; (7), foreign bodies in the alimentary canal; (8), biliary calculi; (9), prostatic calculi; (10), calcareous cyst of the kidney; (11), papilloma of the skin in the lumbar region; (12), appendicular concretions including enteroliths must also be considered. The presence of bismuth and iron salts taken internally can be eliminated by proper preparation of the patient. Phleboliths are usually of round or oval shape and their

image cannot be obtained outside of the pelvic area. Calcified lymph nodes, known also as pelvic blotches, are spherical or oval bodies varying in size from  $\frac{1}{8}$  to  $\frac{1}{4}$  of an inch, but appear as concretions in the pelvic wall. Calcareous or calcified glands as a rule are multiple. They are usually spherical, with blurred outlines showing irregular calcifications, and the shadow is more dense in some parts than in others. A gland is nearly always movable, and can be easily shown by taking several röntgenograms at various angles with full compression upon the abdomen, thereby displacing it. A calculus is more typical in shape, pointed, rectangular or oval, of heavier density throughout its entire structure and with a sharp and clearly defined outline. The renal calculus cannot be moved from one position to another. Biliary calculi being located nearer the ventral than the dorsal wall of the abdomen will show more clearly in the röntgenograms when the plate is placed upon the abdomen, and of course for the same reason renal calculus shows more distinctly when it is placed on the back. In renal calculi the shadows cast on the röntgenogram are of uniform density, whereas in biliary calculi the periphery is more dense, but the central portion less so, giving more or less of a ringlike appearance, namely, dense periphery and dark centers. Furthermore multiple biliary calculi most frequently show facets at the surface of contact, and they usually cast shadows resembling a bunch of grapes, while renal calculi are usually distributed in the kidney, and when united do not show facets. Changing the position of the tube will alter the relation of the biliary calculi to the kidney area, while with renal calculi such changes do not take place and the shadows show always in the kidney area; the latter are also nearer to the spinal column than biliary calculi. Appendicular concretions appear as small circular vacuoles within the appendix shadow. When a calculus is suspected in the lower part of the ureter, the above mentioned possible errors must be considered, though in examinations of the right ureter appendicular concretions may be mistaken for calculi. In doubtful cases, however, röntgenoscopic bougies are helpful in confirming the diagnosis. In reno- and uro-röntgenography the same as in röntgenography of the thoracic and gastrointestinal organs, rapid stereoröntgenography is of value especially in the examination of the bladder. It aids not only in giving length and breadth, but also depth, and its proper relation with the pelvic cavity. By this means we can definitely say whether or not we are dealing with a vesical calculus. In cases presenting gastric symptoms commonly termed dyspepsia, author has found a renal calculus to be present, although no definite symptoms suggesting it were found. In other patients presenting a dull sense of oppression in the right iliac region, which may be mistaken for appendicular affection, it has

frequently proved to be a calculus. Another class of cases presenting chronic epididymitis of several years' standing, which becomes aggravated on exertion, has found to have been due to, or at least complicated by, the presence of a calculus, or calculi in the kidney on the same side.

SACHS.

**X-Ray Efficiency in Bladder Diagnosis**—G. S. PETERKIN, *Interstate Med. Jour.*, Aug., 1916.

The employment of the X-ray in bladder diagnosis offers the following practical advantages: 1. The obtaining of evidence of pathological phenomena often unsuspected and otherwise undemonstrable. 2. That mediums of low specific gravity, as air or oxygen, when the bladder is well distended, will give radiographic evidence of small and otherwise undemonstrable foreign bodies in the bladder, and sometimes of tumors, especially when employed with abdominal position advised. 3. That mediums of high specific gravity, as silver iodid, give evidence as to position, form, size, capacity of the bladder and the presence of abnormalities, as sacculations, etc. For instance, bladder filled with silver iodid will give outline and size of large bladder tumors. Bladder filled, then emptied of silver iodid, will often show size of ulcerations and small irregular growths, as papillomata, due to the iodid adhering to the rough surface. In females it will demonstrate the presence of bladder adhesions, and the pressure on same of tumors and other abdominal organs external to the bladder. The bladder filled with silver iodid will give position of bladder in hernia operations, especially in children. In women with pelvic ptosis, it will demonstrate not only the presence of cystocele, but also the amount of residual urine, etc. 4. The X-ray will give exact data as to the size and number of calculi; information as to the nucleus of stone, if a foreign body—a factor which if not known might complicate removal; presence of calculi behind prostate and in sacculations, in the mouth of the ureter or in that portion of the ureter which runs through the bladder wall—positions in which they could not be localized either by sound or cystoscope. 5. In cases of hemorrhage, pronounced inflammation, diminished bladder capacity, etc., conditions which obscure the cystoscopic view, radiography will often give very useful information; especially is this true in stricture, prostatic hypertrophy, etc. In author's practice it is the rule that radiographs be taken in cases of prostatic hypertrophy, not only prior to operation, but subsequent thereto, in order to ascertain the effect of operation in correcting pathological conditions, especially the mechanical ones of general trabeculation of bladder, sacculations, etc. 6. A series of X-ray pictures gives positive demonstrable evidence as to the progress of disease, effects of operation, etc., which is a cystoscopic impossibility.

WESTERN.

**Diverticulum of the Urinary Bladder**—W. E. LOWER, *International Clinics*, Vol. IV, Series 26.

Frequent urination, with pus in the urine and with inability to completely empty the bladder, is one of the most common signs of the presence of a diverticulum. In case of a large diverticulum, there is often a sudden filling of the bladder immediately after it has been apparently emptied. Even catheterization may not result in the withdrawal of much urine, since the catheter may not enter the orifice of the sac. Another frequent symptom is the passing of clear urine immediately followed by that with more or less pus. Occasionally there is a burning or stinging sensation during or after micturition. Hematuria frequently occurs also. In most cases, this condition does not cause any special annoyance until infection and cystitis develop, when the patient seeks advice because of the pain which accompanies these conditions. In author's series, the average duration of the symptoms was 7 years. If the diverticulum has existed for some time, the kidneys may become infected, and pyelitis, pyonephrosis or hydronephrosis may develop. If the diverticulum becomes infected, the urine, even after repeated irrigations, is cloudy and in a great many cases very fetid. In case the diverticulum is very large, a definite tumor may be detected on rectal or vaginal examination or even by abdominal palpation. In nearly every case, there will be found some complicating or causative urinary obstruction. Often, and especially in cases of elderly men with prostatic obstruction, the symptoms of the diverticulum may be entirely overshadowed by those of the prostatic condition. The presence of a diverticulum can nearly always be determined by the cystoscope in the hands of a person experienced in its use. The opening into the diverticulum is sometimes so very small that it looks like a mere dark speck in the field. This apparently insignificant speck, however, may be the opening into a very large sac. If more fluid can be injected into the bladder than can be immediately drawn out again, one should suspect a diverticulum at once. If the presence of a diverticulum is evident or suspected, the diagnosis may be made certain, and the size and exact location of the sac determined by the aid of collargol and the X-ray. A posterior sac may be overlooked by an anterior-posterior picture, but it can generally be detected by taking a picture at an angle. This seems to be a more exact method than to measure the size of the sac with a ureteral catheter. Kelly and Burnam recommend injecting into the diverticulum a heavy bismuth emulsion, and into the bladder a two or three per cent. emulsion of iodid of silver or some other solution of less specific gravity, then taking a radiograph of the whole. The Brown-Osgood method has also proved very successful in some cases. This consists of taking the X-ray picture after a shadow

or stiletted catheter has been pushed up until it coils into the cavity. In many cases the diverticulum contains calculi. Multiple diverticula are not at all rare. In 9 of author's cases, 2 or more pouches can be plainly seen in the X-ray pictures, one case presenting four distinct sacs, and 2 showing numerous shallow diverticula.

SACHS.

**Sexual Disturbances and Functional Neuroses of the Male Genitourinary**

**Apparatus**—D. A. SINCLAIR, *Urologic and Cutaneous Rev.*, No. 12, 1916.

The dribbling of a few drops of urine, so frequently complained of, after the completion of the urinary act, is a condition which exists in every living man more or less. It is due to the physical law of capillarity, has always existed and always will exist, but the neurasthenic discovers it accidentally, thinks it of recent occurrence, and a symptom of weakness of his genital organ. After the normal urinary act is completed, the few drops which remain in the canal must be gotten rid of by shaking the penis. Author satisfies his skeptical patients by an actual demonstration with a long glass pipette filled with water. When the finger is removed from the proximal end of the pipette, the water runs out, but a few drops are held by capillarity, and must be displaced by shaking the pipette. This demonstration invariably convinces them more firmly than an hour's explanation. Phosphaturia, or the precipitation of phosphates in the urine, is not infrequently met with in men of nervous temperament, and is generally associated with some digestive disturbance, or is due to the ingestion of foods, rich in phosphates and to drugs, which reduce the normal acidity of the urine, rendering it weakly acid, neutral or alkaline, so that the normal amount of phosphates ordinarily held in solution, is precipitated. The appearance of this deposit is not regular, depending as it does on the above named conditions, but when present may attract the patient's attention and is usually interpreted by him as evidence of kidney disease or loss of seminal fluid. A few drops of acetic acid, added to the specimen, clears up the cloud and convinces the patient of the correctness of our diagnosis, which we make and tell him of before the demonstration. Correcting the diet and the administration of dilute hydrochloric acid, fifteen to twenty drops in water after meals, will cause a clear urine to be passed. It is a matter of common experience to hear patients, men and women, complain of the odor, color and general appearance of their urine. They have acquired the habit of urinating in a vessel during the night and examining it the following morning. Although these patients are sensible of the fact that fresh milk will curdle and sour after long exposure to the ordinary room temperature, and that meat will decay under similar conditions, they make no allowance for the urine

following the same natural law, even though it be passed into a dirty receptacle. Variety in color and quantity of the urine depend on the amount of fluids imbibed; the amount of exercise taken, and the season of the year determine the activity of the sweat glands, and hence the amount and color of the urine. A characteristic odor is noticeably present, following the ingestion of drugs and certain foods, such as turpentine or asparagus. The subject of venereal diseases and the sexual relation is one, which we, as men, know to be a common and favorite topic for everlasting discussion amongst the laity; and so it will be as long as the human race supplies new generations. In the club, café or restaurant, over the glass of wine or beer, the young man tries to discover Nature's law, and when we, as physicians, consider the difficulty in satisfactorily explaining even a few of the infinite causes of the defects that human flesh is heir to, we can the more easily understand how the green youth, with little or no knowledge of the physiology of the normal genito-urinary apparatus, can be led astray in the discussion of these sexual disturbances, the causes for which may appear so simple to us. Explanations along rational lines in harmony with the laws of Nature, will solve many a problem, apparently so perplexing to the patient.

SACHS.

**Present Status of Urological Diagnosis**—J. H. CUNNINGHAM, Bull. Dep. Surg., Harvard Med. School.

Graduates of more than 10 years ago will recollect that the presence of pus and blood in the urine was considered to be, at that time, a sign of a serious disturbance within the urinary system, yet the differential diagnosis depended solely upon urinalysis, the general manifestations, and the employment of palpation, percussion and sounding of the bladder for stone; and unless a tumor or tenderness was demonstrated in the kidney region or a stone in the bladder the patient was treated symptomatically. Today the methods of arriving at a diagnosis are quite different. The patient with pus or blood in the urine, or obscure symptoms referable to the urinary system, receives not only the older methods of diagnosis, but by the employment of the cystoscope, the prostate and bladder are inspected and lesions here located are accurately determined. By passing catheters to each kidney, and thus collecting the urine from each organ separately, it is possible to demonstrate beyond question the origin of the abnormal elements as in one or the other organ; and the secretions from each kidney may be subjected to urinalysis and bacteriological study. Not only will important information be obtained by the gross appearance of the separate urines, urinalysis and cryoscopy of the separated urines; but by the subcutaneous or intravenous injections of certain dyes, the func-

tional activity or efficiency of each kidney may be accurately determined by estimating the capability of each organ to eliminate the dye. By these tests it may be decided whether or not there will remain enough renal tissue to sustain life if one kidney is removed; and the data obtained from these functional tests have, moreover, served to determine whether any operation, renal or otherwise, should be undertaken; and to decide whether a renal operation should be a nephrectomy or a nephrotomy. These functional tests have served, also, as an important factor in the understanding of the degree of nephritis from the medical standpoint, and are generally employed in estimating the degree of renal impairment in the consideration of prostatectomy. Röntgen-ray examinations of the urinary system are of value in demonstrating the presence of stone, in outlining the shape and position of the kidney; and in connection with ureter catheterization the internal arrangement of the kidney may be depicted by distending the renal cavity with a shadow-casting solution through the ureter catheter. This method of examination has proved of value in estimating the capacity of the renal pelvis, its size, shape, and internal arrangement, and in hydro-nephrosis has given the valuable information of whether or not the ureter outlet is so located as to drain the kidney pelvis. By distending the ureter with a shadow-casting fluid through a ureter catheter placed but a short distance up the ureter, its course, kinks, strictures, and dilatations may be demonstrated. By this method or by the passage of shadow-casting ureteral catheters into the ureters, shadows resembling ureteral calculi may be determined to be in the ureter or not.

SACHS.

#### FEMALE ORGANS OF GENERATION—PREGNANCY— PARTURITION—INFANTS

**Non-Protein Nitrogen and Urea in the Maternal and the Fetal Blood at the Time of Birth**—J. M. SLEMONS and W. H. MORRISS, Johns Hopkins Hosp. Bull., Dec., 1916.

In 35 normal obstetric patients at the time of birth the average rest-nitrogen in the maternal blood was 25.2 mg. per 100 c.c.; in the fetal blood the average was 24.9 mg. In 16 normal patients the average quantity of urea-nitrogen in the maternal blood was 10.5 mg. per 100 c.c.; in the fetal blood the average was 10.4 mg. The urea-nitrogen represented 44 per cent. of the rest-nitrogen in the maternal and 45 per cent. in the fetal blood. The same concentration of urea in both circulations indicates that this substance passes through the placenta by diffusion. Complications accompanied by an increase of urea in the maternal blood—toxemias of pregnancy, syphilis, de-

Bull.

compensated heart lesions, and others are also attended with a corresponding increase in the fetal blood-urea. SACHS.

**Acute Renal Infection in Pregnancy and Puerperium**—S. H. HARRIS, Med. Jour. Australia, Oct. 7, 1916.

The right kidney was found to be involved in all of the 32 cases of pyelitis gravidarum in which a cystoscopic examination was made. In every case a pure culture of *Bacillus coli communis* was obtained from the catheterized urine from the renal pelvis. Author is of the opinion that pyuria and pain confined to the left side are due to other causes than pyelitis gravidarum and that a pyuria in a case of pregnancy which is associated with other organisms in the renal pelvis than the *Bacillus coli communis* is probably due to some other cause than pyelitis gravidarum. SACHS.

**Rare Cases of Early Eclampsia in Tubal Pregnancy**—F. EBELER, Zentralblatt f. Gynäkologie, 1916, No. 43.

Early eclampsias occur very rarely. The majority of eclampsias ensue during the second half of pregnancy. Author's case was a primipara, 23 years old, with ruptured tubal pregnancy. Laparotomy was performed which was followed by eclampsia. Stroganoff's treatment availed nothing, and patient died. The extra-uterine pregnancy was due to salpingitis nodosa. MILL.

**Difficult Labor Obstructed by a Contraction Ring**—C. WHITE, Brit. Med. Jour., Dec. 2, 1916.

A contraction ring obstructing labor must be differentiated from a retraction ring. A contraction ring is a localized thickening of the wall of the uterus due to the contraction of the circular fibers over a point of slight resistance, most frequently over a depression in the child's outline or below the presenting part. A retraction ring is that part of the internal muscle at the junction of the thinned lower uterine segment with the thick retracted upper uterine segment. The uterine wall at the site of the contraction ring will be thicker than it is either above or below, while the uterine wall above the retraction ring will be much thicker than it is below it. The uterine wall below a contraction ring is not thinned or over-distended, which is the converse in case of a retraction ring. The presenting part of the child in the case of a contraction ring is not forcibly driven into the pelvis as in a retraction ring case. In the former condition of the child may be wholly or mainly above the contraction ring, while in the latter condition part of the child must be below the retraction ring. SACHS.



**Obstetric Paralysis**—J. W. SEVER, *Am. Jour. Dis. Child.*, Dec., 1916.

Obstetric paralysis is due to a stretching or tearing of the cervical roots of the plexus brachialis. It occurs in boys as frequently as in girls. It occurs more often on the right than on the left side. The upper arm type is more frequent than the lower arm type. It affects both arms very infrequently. It is practically always associated with a difficult labor, in which ether and forceps have been used and force has been applied. Not uncommonly is the baby asphyxiated. Head presentations show the larger percentage of occurrences of both types of cases. It may rarely be associated with a fracture of the clavicle, but is not the result of a fractured humerus or a dislocated shoulder joint. The prognosis for a useful arm is good in the upper arm type and bad in the lower arm type.

SACHS.

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HEINRICH STERN, M.D., LL.D., F.A.C.P.  
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## **Special Articles**

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### DYSPITUITARISM

REPORT OF CASES OF DISORDER OF THE PITUITARY GLAND, OCCURRING  
IN THE PRE-ADOLESCENT PERIOD, AND DIFFERING WIDELY  
IN CLINICAL MANIFESTATIONS

By GEORGE A. MOLEEN

Denver, Colorado

The pituitary gland or hypophysis, although one of the smallest of the glands of internal secretion is, as its function becomes better understood, assuming a position of great physiological importance. Its relationship to disturbances in body growth and development especially have revealed the significance to be attached to this apparently unimportant structure.

Since the time Marie and Marinesco, in 1889, were able to attribute symptoms of acromegaly to tumor, and Babinski and Fröhlich showed that adiposity and sexual infantilism were to be associated with the same hitherto neglected gland, a great variety of conditions of over and under growth have been found to be dependent upon alterations in its secretion. The systematic research of Harvey Cushing, by means of animal experiments, surgical procedures and organotherapy, has placed so much material of value before us, helping as it has to unravel the mystery of the hypophysis, as to inseparably link his name with the subject.

Dyspituitarism is a general term under which are grouped the various disorders of secretion of the gland; hyperpituitarism, or over-activity of the gland with excessive secretion; hypopituitarism,

or under-activity with lessened secretion and possibly a combination, since there are conditions which at the outset may be due to over-activity and later become associated with symptoms characteristic of insufficient function of the gland.

It is apparent that such quantitative changes in the secretion may give rise to manifestations which differ widely and seem at times to be opposed; and yet these become more complex when viewed in the light of more recent conceptions of the rôle played by different portions of the same structure. The *pars anterior*, besides its seeming relation to other ductless glands, is more especially concerned in skeletal growth, while the posterior lobe is presumed to have more intimate relation to the processes of tissue metabolism—*i. e.*, when diminished, causing a marked deposition of fat—as well as the activity of the renal and vascular systems. With this in mind, it is obvious that combinations of inactivity of the posterior with increased function of the anterior, or in reverse order, or complete depression or acceleration may give rise to a great variation in the clinical syndrome, and such in fact seems to be the case.

It is interesting to note since this paper was written that Chauvet (*L'Infantilisme*, par Stephen Chauvet, Ed. Maloine, Paris) in his monograph on hypophyseal infantilism has given the most complete summary of the manifestations, first considering the symptom groups or syndromes as follows: Nanism; infantilism of the Briesaud type, the type of Lorain; ateliosis, juvenilism, femininism, eunuchism, genitodystrophic geroderma, reversible infantilism and gigantism with infantilism. Among the morbid disturbances to be associated with a lesion of the hypophysis are mentioned—pituitary pain, olfactory disturbances, visual disturbances, enlargements of the sella turcica, disturbances of adjacent structures, adiposity, disturbances of carbohydrate metabolism, glycosuria, polyuria, polydipsia, thermic disturbances, alterations in the blood pressure, somnolence, etc.

Finally, pertaining to the gland itself, the following four general divisions in accordance with the portion of the structure involved are given:

First, the syndrome of the posterior lobe—the adiposo-genital type of Fröhlich.

Second, a syndrome of the *pars intermedia* of Herring, characterized by polydipsia and polyuria.

Third, the syndrome of hyperpituitarism of the anterior lobe, characterized by acromegaly and gigantism.

Fourth, the syndrome of hypopituitarism of the anterior lobe—hypophyseal infantilism.

The case reports that follow deal with types which while not unusual in a general way, they emphasize some previous conceptions and present peculiarities of sufficient interest to justify their being placed on record.

CASE I. Pre-adolescent dyspituitarism with optic atrophy of right



CASE I.  
Showing infantile stature;  
sexual underdevelopment and  
ocular deviation.

eye and temporal hemianopsia of the left; undergrowth; sexual infantilism without adiposity (type of Lorain); ossification of epiphysial cartilages, incomplete or wanting; corporeal hypotrichosis. Enlargement of sella turcica; probably tumor.

R. L. B. Aged 26 years; referred by Dr. A. J. Simpson; was first observed February 12, 1913. No occupation. Born in Min-



CASE I.

Showing the enlargement of the sella turcica with area occupied by shadow probably of tumor.

neapolis and has been in Colorado fifteen years, but not on account of health. Resides at Longmont, Colorado.

Family history. Mother's condition unknown. Father died of unknown cause. A brother is in good health and development. Little is known of antecedents, character of birth, etc. The patient has been an adopted child since four years of age and since that time has enjoyed good health.

Previous history. No illnesses of childhood other than mumps and scarlet fever at thirteen years of age; the latter was not severe enough to require going to bed, however, nephritis followed a month later and was accompanied by a profuse nasal discharge, during

which a mass that looked like the "core of a boil" came away from the nose. There was severe headache at this time, from which he later recovered and there have been no evidences of kidney disturbance since. After this attack it was noticed that he did not develop; in fact, he was lighter in weight than before, while his height had remained about the same as at that time. There had



CASE I .

Showing the incomplete ossification in the hands.

been attacks of headache with digestive disturbance, which were obstinate, but no vomiting since the above-mentioned sickness. There had been no loss of muscular power at any time.

About eight years ago without warning he became rigid, lost consciousness and upon recovery experienced a feeling of fatigue. Another convulsion three months later, similar to the other, which was attributed to digestive disturbance and without loss of conscious-



ness. There had been none since. There was disturbance of vision at the time of the attack of nephritis, but recovered quite fully later. The right eye began to fail in vision six months later, however, and progressed slowly until about eight years ago, when sight was completely lost; there was no recollection of double vision. Prior to the loss of vision of the right eye, it was noticed he was unable to see things on "either side of him" and only when they were placed directly in front of him. With this loss of sight the right eye began to turn inward. For the past two years there has been a failure of vision in the left eye.

About six years ago an attack of facial erysipelas was followed by recovery. Otherwise he had been in good health, but there had been no increase in height since the thirteenth year and some diminution in weight.

Intellectually he had progressed and his memory had been excellent. He attended school up to the seventh grade and later went to a school for the blind for two and one-half years. Educationally he progressed in arithmetic as far as algebra, finished physical geography and was at that time in rhetoric and English composition.

Present history. The complaint at that time was loss of vision, outside of the diminished stature. He denied the use of alcohol and tobacco, and had never indulged in sexual intercourse.

Appetite was generally good. Bowels had always been constipated; he slept well.

Examination on February 12 and 16, 1913, showed a symmetrically undersized male of 142 cm. (56 inches) in height, weighing 65 pounds.

The head was proportionate in size to the remainder of the body; the vertex rather high, forehead slightly receding and very slight prognathism. The ears were well formed, lobuled and symmetrically placed.

The skin of the face presented a dry wrinkled appearance, the forehead being furrowed and the eyes wrinkled and there were radiating creases at the angles of the mouth.

The skin over the trunk, and particularly over the extremities below the knees, was dry and scaly.

The general color was quite good and there seemed to be but little superficial fat, with the exception of the area over the pubes

there seemed to be slight accumulation which appeared disproportionate.

The hair of the head was plentiful, but there was practically no hair on the face, arm-pits or pubes. Patient had never shaved, except on a few occasions to remove a few isolated hairs.

The teeth were well formed, not unusually spaced, showing a number of fillings, but no marginal cusps.

The palatine arch was broad and well formed and was proportionate in size to the rest of the head and body. The circumference of the head was 50.5 cm.; from the subnasal point to the external occipital protuberance, 31 cm.; bi-aural diameter, 34 cm. From the extremes of finger tips, 141 cm., the right 71 cm., the left 70 cm. The hands measured 16 cm. on either side. The fingers tapered and the nails were normal. Dynamometer, R. 130, L. 120. Patient was right-handed. From the perineum to the sole of feet (standing), 67 cm. Pupillary distance, 50 cm. (evident convergence); between external canthi, 80 cm. The brow is wrinkled evenly. The tongue was protruded in the middle line, the mouth retracted equally, and the palate was drawn equally on phonation. The eyes were closed with equal firmness. Muscular action throughout the body was well performed and with good power. No muscular atrophies were observed. Associated movements were well executed, and no abnormality of movement of the lower extremities could be made out. There was no impairment of gait and station, and no tremor in any part of the body.

The heart was normal in outline, no adventitious sounds over the valve areas; regular in rhythm; eighty in rate and the radial pulse was of good quality and 115 mm. in systolic pressure—later estimations were recorded as 92 mm. on March 23, and 105 mm., on December 24 of the same year following the administration of pituitary substance. The lungs were normal and the abdominal organs were apparently normal.

The sexual development was much diminished; penis, small; foreskin, elongated; no paraphymosis. Testes, distended, soft and small, though proportionate with the genitalia.

Reflexes. All of the deep reflexes were present, about normal in degree and equal; there were no clonusus.

Superficial reflexes. Plantars were both flexor. Compression

of calf, R. flexion, extension of great toe; L. flexion followed by extension. Cremasteric, R. and L. prompt. Oppenheim, flexor on either side. Upper and lower abdominals were prompt and equal. Epigastric, present. Cervico-pupillary, R. and L. present.

Special senses. General sensation: Tactile and pain senses were prompt. Pressure was accurately recognized. Postural was present. Localization was accurate and prompt. Temperature was present. Stereognostic sense was present in either hand.

Eyes. All external ocular motion was present, except for a weakness of the right external rectus; on extreme effort this could be moved, but was not rotated outward in association; in convergence it was lagging as well. There was no nystagmus in any direction. Pupils: 4 mm., the left responded but slightly, if at all. There was more response apparent in the consensual influence of the left pupil, particularly when light was thrown on the temporal side of the retina of the right eye, when a slight response of the pupil of the left was observed. This did not occur when light was thrown onto the nasal half of the right eye. The direct response under the same conditions was barely perceptible. Fundi: Right, pale; vessels, attenuated and moderately sclerotic; some perivascular exudate; no hemorrhage; disk, sharply outlined, chalky-white and no nutrient capillaries were present. The left was the same as the right, but not so marked in degree; the outline was sharper in the right than in the left eye. Both were seen with  $\odot$  aperture. Fields of vision taken by Dr. Bane: Right, nil. Left temporal field was more amblyopic; this field was much contracted and almost limited to central vision (see chart). Vision: Right, nil, no perception to light. Left,  $1/240$ —; fingers were counted at twenty-four inches.

Hearing. (Watch =  $24''$ .) Right,  $30/24$ ; left,  $30/24$ . Tuning fork was heard best in the closed ear. Aerial conduction was greater than bone in either ear.

Taste and smell were both present and prompt.

Clinical diagnosis. Pituitary insufficiency. Tumor of the hypophysis.

CASE II. Pre-adolescent, dyspituitarism—insufficiency—type of Fröhlich; probably secondary to cerebral tumor; mental deficiency; nystagmus; bilateral optic atrophy with concentric contraction of the fields of vision; pronounced corporeal adiposity; sexual precocity.

M. S. Female. Aged 9 years, referred by Dr. A. H. Williams; was first observed October 8, 1915. Patient was born in Colorado and lived here since.

Family history. Revealed the mother and father in good health;



CASE II.

Showing the adiposity; marked development of the breasts, with the secondary characteristics.

a brother died in infancy, and a sister and brother were living in good health.

Previous history. Birth was normal and without injury; breast-nursed and infantile life was normal without spasms and no dysenteries. Speech began at one year and walking at eighteen months. On account of polyuria, she was believed to have been diabetic. Measles at two years and whooping cough at five years. When

about four years of age, while manifesting the polyuria and about ten o'clock in the morning, while lying in bed, she was observed to draw the mouth to the right and the head to the left and the right eye-lid was somewhat drooped. In about a week improvement began, but the drawing remained more or less since that time. There had been some vomiting without nausea, but there had been no complaint of headache. About two weeks following this she was able to be about, but the face had never been straight. She was unable to speak and has been mentally dull since this time.

There was no absolute loss of power in the right arm or leg, and the mother is quite sure that they were moved on the day following the attack, but that they have been somewhat weaker than the left.

About two years ago the great toe of the right foot began to turn upward and the power in the right leg had failed gradually since that time.

About a year ago she was noticed to be developing unusually; the breasts began to fill out and pubic hair, as well as that in the armpits began to appear. Menstruation appeared five months ago with some pain and a flow three days in duration. There had been considerable increase in weight, more especially was it apparent in the trunk.

At the present time there is progressing loss of power of the right leg, arm and the right side of the face. Increasing mental weakness, more especially noticeable in the retardation of mind. Large quantities of urine are passed without sugar. Vomiting at times when she attempts to satisfy the excessive thirst. There is no nausea and but little wrenching. Dizziness is experienced with subjective vertigo toward the right side. Occasional frontal headache. Appetite, fair. Bowels, sluggish or not regular. Sleep, quite good.

The examination, October 11, 1915, revealed an unusually well developed girl with striking precocity of sexual attributes with noticeable degree of sub-cuticular fat. The breasts were quite large, while the nipples were small and that of the left was retracted. The abdomen showed considerable adiposity and the trunk generally more so than the extremities.

The head was rather larger than ordinary, considering the age, but there was no change in the direct percussion note. She was somewhat anemic in color.

The hands were quite small and the right somewhat smaller than the left and the fingers showed marked tapering. There was considerable pubic hair and the axillae also were well supplied. The skin of the right extremities, and the right side generally, was cooler to the touch than the left. The right leg was somewhat smaller below the knee than the left, while little difference was noticed in the thigh. The position of the foot on the right side was equinovarus. The right eye-lid drooped. The right side of the face showed less motion than the left. The tongue was protruded in the middle line. The mouth was retracted more to the left. On phonation, the palate appeared to be drawn more to the right side. Speech was clear in enunciation, but slow in utterance. Cerebration was noticeably retarded. Associated movements were somewhat impaired in the upper extremity and on voluntary movement a slow ataxic tremor was to be observed in both hands, but more pronounced in the right; this was absent while at rest and became most marked just at the completion of an act.

She was able to stand on the left leg, but with considerable unsteadiness, while the right was too much weakened to do so.

Reflexes. Knee jerk, R. slightly increased; L. normal. Ankle clonus, R. and L. absent. Tendo Achilles, R. slightly increased; L. present. Deep reflexes of the forearm, R. increased; L. present. Biceps, triceps and deltoid, all slightly increased on the right side. Supinator jerk, R. prompt; L. present. Masseter, R. and L. absent. Jaw jerk, slightly increased.

Superficial reflexes. Plantar, R. present and frankly extensor in type; L. flexion. Compression of calf, R. no action; L. flexion. Oppenheim, R. positive; L. negative. Lower abdominals, R. absent; L. absent. Upper abdominals, R. and L. absent. Epigastric, absent. Cervico-pupillary, R. and L. present.

Special senses. General sensation, including tactile, pain, pressure, postural and localization senses were present and equal throughout. Temperature sense was fairly accurate. Stereognostic sense was present in both hands.

Eyes. All external ocular motion was present, but nystagmus was evident to a moderate degree on forward fixation. Lateral nystagmus was to be seen in either eye. Rotation in either direction increased, while the greatest augmentation was present upon rota-

tion to the left side. Fundi: Both disks appeared atrophic; the margins were not clearly outlined and the atrophy apparently extended into the retina giving one the impression of a type secondary to swelling or post-neuritic. The fields of vision were apparently contracted concentrically. Vision could not be accurately estimated.

Hearing. (Watch = 24".) R. 24/24; L. 24/24. Tuning fork was heard best in the closed ear. Aerial conduction was greater than bone in either ear.

Taste. Was not examined. Smell: Was prompt on either side.

Clinical diagnosis. Hypopituitarism, probably associated with cerebral tumor in the left subcortex.

Unfortunately through an oversight the röntgenograph of this case was not obtained, but the clinical symptoms indicate the presence of tumor with secondary pressure upon the hypophysis so clearly that there can be little doubt as to the diagnosis and the fact that the tumor has existed since early childhood renders the type of dyspituitarism as pre-adolescent and in conformity with the descriptions of Fröhlich and others.

CASE III. Pre-adolescent dyspituitarism; probably moderate hypersecretion, acromegalic in type; characteristic enlargement of the interphalangeal joints; slight, but distinct bony enlargement of the face; moderate thickness of the skin with coarseness of hair; no changes in general body development, except the scaphoid type of scapulæ; no sexual changes.

F. A. J. Aged 18 years; occupied in newspaper work; was first observed April 19, 1914.

Family history. Revealed father and mother in good health; two brothers dead; one of pneumonia and other of consumption; two sisters were in good health, one sister died of exophthalmic goiter since the record was taken; negative including nervous and mental diseases.

Previous history. Whooping cough, measles and mumps as a child. There have been no other sicknesses, and gonorrhea and syphilis were denied. No alcoholic liquors have been taken. There had been no injury of consequence.

At about thirteen, a stiffness in the hands was noticed, especially in the mornings, followed by a slight enlargement of the second interphalangeal joints. At about the same time the metatarso-



CASE III  
Showing roentgenograph of the head.



CASE III.  
Showing enlargements of the joints in the feet.



phalangeal joints of the great toes became enlarged. There had been no pain in any of these joints, except occasionally in the feet from pressure of the shoe.



**CASE III.**

Showing the scaphoid type of scapula.

Present history. Complaint was limited to the condition of the joints, otherwise in good health. There had been no cough, no headache, no dizziness, and no vomiting. Appetite was good; bowels, regular and sleep good.

Examination showed a boy of spare development; rather coarse featured and of fairly good, though tawny color; five feet, eight inches in height, and weighing one hundred and thirty-three pounds. All of the fingers of both hands showed a decided enlargement of the second interphalangeal joints without palpable enlargement of the distal phalanx. The metacarpals of the phalangeal joints were



CASE III.

Showing typical hyperostosis of the distal phalanges.

but slightly increased in size. A great enlargement of the metatarso-phalangeal joint of the great toe of either foot was present. The enlargement appeared and conveyed a sense of edema and seemed to fluctuate. Aspiration showed a viscid, clear fluid, which was so thick that only a small portion could be removed.

Cerebration was good. Memory was good for recent and remote events. Tongue was protruded mesially without tremor. Mouth was retracted evenly. Palate was equally drawn. Upper and lower facial muscles acted well. Muscular power in the grasp was some-

what diminished owing to the stiffness in hands and enlargement of joints. Associated movements of the hands were well executed. The lower extremities on either side showed good power. Gait and station were without defect, on movement or co-ordination. There was no tremor to be seen in any part of the body. Dynamometer, R. 190; L. 120. Patient was right-handed.

All of the deep reflexes were present, equal on the two sides and normal in degree. There were no clonuses. The superficial reflexes were present, equally active and normal in type, the Oppenheim and Gordon reflexes being negative. Of the special senses, no abnor-



CASE III.

Showing the development of the lower jaw and the general contour of the face suggesting acromegaly.

mality in perception of touch or pain was to be made out, and the stereognostic sense was present and accurate in either hand.

Eyes. All muscular movement was present in all directions and there was no nystagmus. Pupils were 2.5 mm., equal and actively responsive to light, distance and convergence. The fundi were normal. The fields of vision showed no alteration to finger testings. Visual acuity, O. D., 20/20; O. S., 20/20.

Hearing. Right and left, 24/24. Aerial conduction was greater than bone in either ear. Tuning fork was heard best in the closed ear.

Hemoglobin, 75 per cent. (Dare).

Blood pressure. Systolic, 110 mm., diastolic, 90 mm.

The chief features presented by this case are the symmetrical enlargements of the interphalangeal joints, and the general appearance assumed by the hands, resembling those which have been termed acromegalic in type. The suspicion of the pituitary in this case is further emphasized by the enlargement of the bones of the face, peculiarities of the development of the jaw and the coarseness



CASE III.

Showing the enlargements of the fingers.

of the skin, as well as the suspected enlargement of the sella turcica as shown in the röntgenogram of the skull.

For the purpose of comparison, a fourth case is to be briefly mentioned, which is an adolescent type of moderate acromegaly in a man forty-nine years of age, whose family history shows tuberculosis on the paternal side, as well as the occurrence of goiter in

the mother, maternal grandmother, a sister and a brother. All were simple enlargements and it is stated that none became active. There was a suspicion of specific infection in the previous history. The



CASE IV.

Showing the obliteration of sella turcica with the occupation of the area by clear shadow.

first symptoms were dizziness, localized at the top of the head, with a sense of vertigo. It is stated that the heart beat rapidly and that there were black flashes before the eyes. The chief complaint now is pressure in the head and beating of the heart with "skips." There were subjective sensations of going forward and occasionally back-

ward. A sense of pressure in ear drums. General condition otherwise good.

The thyroid gland was removed in its entirety from the right and partly from the left side at the beginning of the symptoms about two years prior to his observation in 1915, as a result of the protrusion of the eyes, the rapidity of the pulse and the swelling of the thyroid gland. The protrusion of the eyes disappeared after the operation.

Examination, December 29, 1916, showed a man of six feet, weighing two hundred pounds, of swarthy complexion and plethoric in type. The facial features were conspicuously large and especially



CASE IV.  
Showing definite acromegalic countenance.

the lower jaw. There was slight exophthalmos and the eye-lids lagged somewhat. The pulse was compressible and ninety-six in rate. A fine toxic type of tremor of the hands was present. The face muscles acted equally. The teeth were spaced somewhat more than normal, and the skin was coarse and of a brownish, tawny color. The hair was also coarse in texture. There was no abnormality in the mental condition, other than a tendency to introspection, which accounts for the subjective sensations.

All reflexes were normal and equal in degree. There was no impairment of the sensation. The movements of the eyes were normal. The pupils were equal and actively responsive to light and distance. The fields of vision were not noticeably impaired. Vision, O. D., 21/100; O. S., 21/100, and the patient was a high myopic.

Optic fund: R. seen best with a —16 D, and showed a somewhat hyperemic nerve with patches of choroidal inflammatory remains near the nasal half. The left eye was best seen with a -12D. The retinae were otherwise normal. Hearing was not impaired. Blood pressure, systolic, 130 mm.

The skiagram shows a definite enlargement of the sella turcica and there is a shadow which is suspected to be cast by that of a tumor or cyst in this region.

The case is added as a case of true acromegaly of the adult type, in order that contrast may be had for the alterations of the secretions of the pituitary gland, between the adult and pre-adolescent types. It also indicates a tendency in many cases of a participation of several of the ductless glands in the same case. The association of the thyroid with hyperplasia of the pituitary in this case seems undoubted and commingling of the symptoms of exophthalmic goiter with those of acromegaly are interesting.

The first three cases seem to require no comment and are presented as examples of established types of the effects of the alteration of the secretion of the hypophysis.

In conclusion permit me to say, in no condition has the röntgenograph been in such singular service than in the elucidation of diseases of this gland, and it seems fitting to express my appreciation of the excellence of the work which has been done in the cases mentioned—some of which were taken by the late Dr. Stover, some of the excellent views of Case II, by Drs. Childs and Crosby, and the excellent skiagram of the skull of the case of adult acromegaly is to be credited to Dr. L. W. Storey, of the County Hospital; to all of whom I gratefully acknowledge my appreciation.

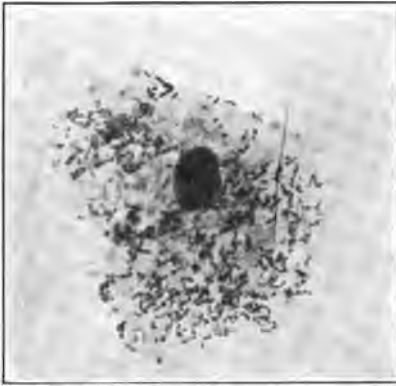
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#### ENDOTHELIAL CELLS—A NORMAL BLOOD CONSTITUENT—A NEW STAINING METHOD FOR MITOCHONDRIA

By FREDERICK PROESCHER AND HARVEY A. SEIL  
Pittsburgh, Pa.

In 1874 Ponfick<sup>1</sup> called attention to the presence of fatty degenerated endothelial cells in the blood stream of patients suffering from recurrent fever. This observation was apparently over-

# THE ARCHIVES OF DIAGNOSIS



**FIG. 1.**  
Endothelial Cell—Human Blood. Methylene Azure. Leitz  $\frac{1}{12}$  Oil Immersion; Ocular 2.



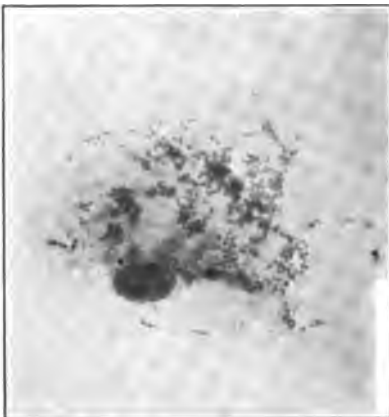
**FIG. 4.**  
Endothelial Cell—Rat Blood. Methylene Azure. Leitz  $\frac{1}{12}$  Oil Immersion; Ocular 2.



**FIG. 2.**  
Endothelial Cell—Horse Blood. Methylene Azure. Leitz  $\frac{1}{12}$  Oil Immersion; Ocular 2.



**FIG. 5.**  
Endothelial Cell—Guinea Pig Blood. Methylene Azure. Leitz  $\frac{1}{12}$  Oil Immersion; Ocular 2.



**FIG. 3.**  
Endothelial Cell—Rabbit Blood. Methylene Azure. Leitz  $\frac{1}{12}$  Oil Immersion; Ocular 2.



**FIG. 6.**  
Endothelial Cell—Pigeon Blood. Methylene Azure. Leitz  $\frac{1}{12}$  Oil Immersion; Ocular 2.

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THE ARCHIVES OF DIAGNOSIS



FIG. 7.  
Endothelial Cells—Human Blood. Methylene Azure. Leitz  
Objective 4; Ocular 2.

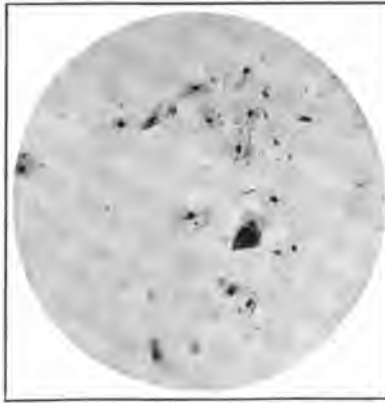


FIG. 8.  
Endothelial Cells—Rabbit Blood. Methy-  
lene Azure. Leitz Objective 4; Ocular 2.

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looked and modern text-books on hematology either do not mention it at all or merely record it as an interesting finding without appreciating its significance. According to Ponfick, the fatty degenerated endothelial cells were exfoliated from the intima of the vessels. In recurrent fever the number of these cells depends upon the intensity of the fatty degeneration of the walls of the vessels. Ponfick furthermore held that the presence of these cells was due entirely to extensive fatty degeneration in recurrent fever, while in other infectious diseases as severe erysipelas, fatty degenerated endothelial cells could not be demonstrated since the fatty degeneration is less intensive and fewer cells are expelled, making their detection in the peripheral blood improbable. He also found "Körnchenzellen," fatty degenerated pulp cells, in venous blood coming from the spleen. In conclusion he also pointed out the possibility of these cells being the cause of embolic disturbances.

One of us (Proescher)<sup>2</sup> has demonstrated the presence of endothelial cells in the peripheral blood of typhus fever patients, particularly about the time of the crisis. Many of these cells contained a small diplococcus which was regarded as the causative agent of the disease. Besides these cocci larger polymorphous structures in the form of granules, rods and filaments were observed. At that time these findings were regarded as typical for typhus fever since these cells were found neither in normal blood nor in the blood of patients suffering from typhoid fever, scarlatina, measles, variola parotitis, etc.

We<sup>3</sup> have recently shown that a large number of endothelial cells occur in the blood of hogs affected with hog cholera. Here as well as in typhus fever fixation with methyl alcohol followed by staining with methylene azure demonstrated endothelial cells. A similar procedure with normal blood did not reveal the presence of endothelial cells.

Inasmuch as methyl alcohol preserves all the elements of the blood smear, it is obvious that an occasional endothelial cell will be masked by the larger number of red and white cells and thus escape detection. In an investigation on vanadium<sup>4</sup>, numerous vanadium salts were prepared and their pharmacological action studied as well as their usefulness as fixing agents upon different tissues, especially the blood. Sodium tetravanadate and sodium

hexavanadate were found to be differential fixatives, dissolving the red and white blood cells and the blood platelets while preserving the endothelial cells and microorganisms. In hog cholera blood, fixation with either of these salts, followed by staining with methylene azure, showed a considerable number of endothelial cells as well as the microorganisms causing the disease.

Control experiments with the blood of normal hogs, fixed in a 5 per cent. aqueous solution of either of the vanadium salts showed endothelial cells as a normal blood constituent. The importance of this finding is apparent and warranted a more extensive investigation. Examinations of the blood of various animals corroborated the original finding and endothelial cells were found to be a normal constituent of the blood of man, horse, mule, sheep, rabbit, rat, mouse, guinea-pig and pigeon. In all instances blood smears were made either from the peripheral or heart blood. In order to prove that the endothelial cells truly circulate in the blood-stream, blood was taken from a horse by venesection of the jugular, and after it flowed freely smears were made excluding any contamination of the blood by endothelial cells set free by attrition.

The air dried blood smears were fixed in a 5 per cent. aqueous solution of either sodium tetravanadate or hexavanadate heated to 60 deg. C. for about five minutes. The cover slips were then washed in running water and floated on a 1 per cent. solution of methylene azure carbonate containing 1 per cent. of phenol. In place of the methylene azure carbonate, carbolfuchsin, Gram or Giemsa solution may be used. The most effective stain is the strongly basic methylene azure carbonate.

In smears stained by the above method with methylene azure carbonate the endothelial cells appear as polygonal, flat or spindle-like cells with a transparent homogeneous or irregularly lumpy protoplasm stained either from a light to a deep blue. The nucleus is stained either a dark or a metachromatic blue and sometimes contains one or two nucleoli. Many cells show regressive changes, loss of mitochondria, an indistinctly stained nucleus and a diffuse staining of the protoplasm. The size of the cells varies from 25 to 75 micra; the nucleus from 5 to 8 micra. The majority of these cells occlude in their protoplasm deep blue granular, rod-shaped or filament-like elements closely resembling microorganisms.

Their size varies from .2 to 7 micra. They occur either isolated, irregularly distributed throughout the protoplasm, or in closely aggregated formations sometimes completely filling the entire protoplasm and may cover the nucleus. They also occur extracellular, near the cell and occasionally distributed over a considerable area. These structures are unquestionably mitochondria (Benda) since they are stained by janus green and diethylsafranin.

In a recent article E. V. Cowdry<sup>5</sup> gave an excellent review of our present knowledge of mitochondria. He showed that mitochondria is no recent discovery and was undoubtedly described by a number of the older authors as Altmann, Flemming, Köllicker, Arnold, Heidenhain, etc. There is still considerable confusion as regards the relation of mitochondria to other protoplasmatic constituents, but recent investigations with modern staining methods, particularly with vital staining, have shown that mitochondria is a well-defined class of cell structures, which must be separated from other cellular granulations to which they bear no relationship. This phase of the question is thoroughly and clearly discussed by Cowdry, and the reader is referred to his article.

Mitochondria can be stained by several dyes, one of which is applicable to vital staining. This latter dye, janus green, is a member of the safranin family. Cowdry has shown that diethylsafranin stains mitochondria directly. The janus green method first described by Michaelis is very delicate. Cowdry found that it stains mitochondria in human lymphocytes in a dilution of 1 to 500,000. The reaction is dependent upon the presence of the diethyl group.

The methylene azure carbonate method described above used in staining the endothelial cells also clearly and sharply stains mitochondria, and demonstrates its presence in endothelial and epithelial cells, in the interstitial cells of the testicle as well as in the brain cells in certain pathological conditions. These structures are also stained vitally by janus green and diethylsafranin. This method is most convenient and rapid for rendering mitochondria visible in smears and will be of value in the study of the changes in mitochondria in pathological conditions. After fixation with sodium hexavanadate, sodium tetravanadate, potassium bichromate, alcoholic bichlorid or ammonium uranictrate the common stains, as Gram and carbolfuchsin stain mitochondria, but not as satisfactory as methylene azure.

Notwithstanding the fact that mitochondria is regarded as a lipoprotein, it is not dissolved by 3 per cent. acetic acid, chloroform, ether, alcohol, tetrachlorethane and 25 per cent. antiformin. These findings are contrary to previous observations, for in sections mito-

chondria was stated to be soluble in acetic acid, alcohol and the immiscible fat solvents. The close similarity of mitochondria to microorganisms renders the differentiation between these structures difficult. There is no doubt that some of the structures described by Prowazek as chlamydozoa, and cell inclusions are mitochondria.

It is logical to assume that the endothelial cells of the vascular as well as of the lymphatic system like the epithelial cells of the pleural, peritoneal and pericardial cavities undergo continuous desquamation. It is difficult to decide which of these originate from the vascular and which from the lymphatic system. It is probable that the majority have their origin in the intima of the blood vessels, since the lymph glands act as a filter, taking up the cells coming from the peripheral lymph vessels. The intima of the blood vessels is exposed to more sudden changes than the cells of any other organ. Mechanical, chemical and osmotic changes are common under normal conditions. If the desquamated endothelial cells undergo autolysis in the blood stream or are removed by the spleen or other organ is not known. However from the appearance of the stained cells which show all forms of regressive metamorphosis, loss of mitochondria, lumpy degeneration of the protoplasm, karyorrhexis of the nucleus, it seems very likely that many of the cells are destroyed in the blood stream. If the number of the desquamated endothelial cells in a given volume of blood have any relationship to changes in the intima under normal or pathological conditions cannot be decided, since no method for determining endothelial cells in the blood stream is at present available. If the regressive changes in the cells themselves will be of diagnostic value must be determined by further investigations.

In 1910<sup>6</sup> Patella called attention to his theory that the endothelial cells of the blood vessels are the source of the large mononuclear cells of the blood stream. According to this author the large mononuclear leukocytes have a great similarity to endothelial cells. In his opinion these leukocytes are metamorphosed endothelial cells. He also describes cellular elements which have the appearance of squamous epithelial cells. From their morphology and arrangement he pronounces them endothelial cells. The mononucleosis in pneumonia, typhoid fever and other exanthematic diseases, according to Patella, is the result of an infectious en-

endarteritis caused by an increased desquamation of endothelial cells. The mononucleosis produced by trauma upon the vessel wall as by intensive massage or clonic convulsions (chorea) should also be caused by an abnormal exfoliation of endothelial cells. He believes that the intensity of the mononucleosis is a very valuable indicator of the condition of the intima in infectious diseases. His theory is not accepted by the majority of the hematologists. Pappenheim refutes this theory and holds that the endothelial cells seen by Patella are accidental contaminations from the epithelial covering of the skin. He further states that the mononucleosis observed in malaria is not the result of an endarteritis, since no changes in the intima caused by this disease are noted. Therefore, the mononuclear leukocytes must be produced elsewhere.

Our observations refute Patella's theory, since a large number of endothelial cells are found in normal blood without an increase of the mononuclear cells. Of course the origin of the mononuclear leukocytes cannot be disputed, since all blood cells originate in the vessel wall. It is our belief that the mononuclear leukocytes are not dead metamorphosed endothelial cells, but fully differentiated potent cells of indefinite origin. The morphological habitus of a necrobiotic endothelial cell and a mononuclear leukocyte is markedly different in their protoplasmatic as well as in their nuclear structure. Furthermore, if there existed the relationship described by Patella gradation stages from endothelial cells to mononuclear leukocytes should be found. Such, however, is not the case.

In conclusion, endothelial cells are constantly present in human blood as well as in the blood of various animals. The cells are probably desquamated from the blood vessel wall, but may also have their origin in the lymph vessels and lymph glands. The majority of the cells contain mitochondria showing their recent origin and relative age. They may be destroyed by autolysis in the blood stream, since many cells show a loss of mitochondria and signs of regressive metamorphosis as nuclear disintegration and lumpy degeneration of the protoplasm. The common staining methods do not reveal endothelial cells in normal blood if methyl alcohol or heat is used as a fixative. Under normal conditions it seems that their protoplasm is strongly basophilic and can be made visible only after mordanting with the certain metallic salts followed by

staining with a strong basic dye as methylene azure carbonate. If blood is hemolyzed with distiller water and centrifuged and the sediment stained directly, the cells are made visible, but very imperfectly stained. In pathological conditions some change in the protoplasm must take place since fixation with methyl alcohol followed by staining with methylene azure carbonate renders some visible. All can be stained only when previously mordanted with a suitable metallic salt. Further investigation of the blood in pathological conditions is necessary to determine if the endothelial content can be either qualitatively or quantitatively of diagnostic value, particularly with reference to the condition of the intima of the vascular apparatus. It may also be helpful in the diagnosis of diseases of the lymphatic system if an alteration of the vascular apparatus can be excluded. The findings *intra vitam* should be controlled *post-mortem* in order to afford a sound basis for an exact differential diagnosis. The findings in typhus fever and hog cholera make it probable that the endothelial cells are the primary foci of infection and warrant a closer study of these cells in various infectious diseases of unknown origin such as scarlet fever, measles, yellow fever, etc., and of known origin such as tuberculosis, since they may play an important rôle in disseminating the bacilli (miliary tuberculosis). Furthermore, the pathology of the hemorrhagic diathesis, hemophilia, purpura and scurvy may also be benefited by a similar study.

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## THE DIAGNOSIS OF THE ARTERIAL GROUP

By STEWART R. ROBERTS

Professor of Medicine, Emory University,  
Atlanta, Ga.

Out of the vast amount of research, clinical experience, and literature there is gradually emerging a clinical classification that permits a fairly accurate diagnosis of the sub-divisions of the arteriosclerotic-hypertensive group. The word arteriosclerosis preferred by Aschoff and Benda<sup>1</sup> is probably a better term than arteriosclerosis, though Allbutt would limit it to disease of the intima. Hypotension is not included in the group. Allbutt's<sup>2</sup> classification is largely followed, and to him is due the credit for separating and describing hyperpiesia and decrescent sclerosis.

I. Hyperpiesia. High pressure, hypertonus, or hypertension. High pressure primary, cardiac hypertrophy and arteriosclerosis secondary. Clinical picture of nephritis absent. Causes indefinite. Prolonged strain and high living probable factors.

II. Toxic Type. Arteriosclerosis the chief lesion, with or without high pressure. Causes general, involving infectious diseases, industrial poisons, dissipation with food, drink, and tobacco, physical labor plus other toxins, endogenous poisons, bad tissue chemistry.

III. Renal Type. Arteriosclerosis of renal origin, the granular, contracted kidney, hypertension, cardiac hypertrophy. The clinical symptoms of chronic nephritis of the vascular or chronic interstitial type. Cardio-vascular renal disease.

IV. Decrescent arteriosclerosis. Vessels palpable, tortuous, and even looped, heart not hypertrophied, pressure normal or amplified slightly by age. A fibrous withering of the entire system. A senile change.

A few simple distinctions and definitions are necessary to give the proper conception of the group. Arteriosclerosis is one condition—an anatomical change in the vessel wall; hypertension is quite another condition—a mechanical increase of the fluid pressure within the vessel. Either may exist without the other, or they may occur together. However much morbid anatomy and perverse circulatory mechanics may underlie the group, a clinical classification rests chiefly on clinical signs and symptoms, and clinical rather than



pathological definitions are necessary. The clinical definition sums the clinical picture.

Arteriosclerosis is an increase in the thickness, resistance, and palpability of an artery for the age of the individual. Thicker arteries at sixty mark the wear and tear of three score years, but such arteries at twenty would be distinctly pathological. Hypertension is an increase in the systolic or diastolic pressure, or both, for the age of the individual. A pressure of 120 m.m. at twenty years may rise to the "quasi-normal" (Allbutt) pressure of 150 m.m. at sixty years. *For the age of the individual* is the key to interpretation both of arterial palpability and arterial pressure. Once arteriosclerosis has developed, it remains, though in certain cases further development may be slowed or even prevented. However it is well to remember that the greatest cause of the condition is age, and each individual is every day growing older and thereby through time inviting sclerosis. In certain types pressure likewise tends to rise with age and we allow an amplitude of rise proportionate to age. While in the first type hypertension is the initial evidence and indicates at least a pathological physiology, in the renal type it is distinctly compensatory, and indeed is really a physiological pressure in view of the renal disease. Arteriosclerosis is not of itself directly a cause of cardiac hypertrophy as proved by the decrescent type, but hypertension is of itself a prime cause not only of ventricular hypertrophy—the high pressure hypertrophy of Riesman,<sup>4</sup> but probably indirectly of an hypertrophy of the media of the arteries also,<sup>5</sup> as proved in turn by the hyperpiesia group.

Back of arteriosclerosis and back of hypertension is a motley and ubiquitous tangle of causes whose very number and variability acting usually through a long series of years, each factor with a separate influence varying in different individuals, and all accented by the ever present "time toil, and tension," make it impossible in the present state of our knowledge to state definitely the demarcations of cause or of effect. The two conditions are not more pathological than are the causes that produce them. In addition they are evolutionary in development, unfold far more slowly than the toughest acorn, and even though slow and hesitant, may show haste if life be too stormy and strained or disease and toxins too severe or the tubes too little resistant.

A more detailed discussion of the symptoms characteristic of each group with illustrative cases shows the chief diagnostic differences, and yet always here the shading of one type into another, the gradual bringing of the kidney into the picture in advanced cases of other than the renal type, and the inability often to place the tottering cases in any one group, are all facts to be remembered.

1. *Hyperpiesia*, an initial hypertension usually without known cause, with the gradual development of cardiac hypertrophy and arteriosclerosis without the clinical picture of nephritis. The hypertension probably precedes the sclerosis. After sclerosis has developed the kidney is of the arteriosclerotic type, and is not to be differentiated grossly at autopsy from the kidney of a toxic arteriosclerosis. Hypertension, arteriosclerosis, the arteriosclerotic kidney are probably the renal order. The presence or absence of a few tube casts is not final evidence of an interstitial nephritis. The cardiac hypertrophy is due to the hypertension. The seriousness of the case is usually proportionate to the height of the pressure, particularly the diastolic pressure.

In *hyperpiesia* before sclerosis has developed there are frequently no symptoms. Extreme hypertension is compatible with a feeling of health and buoyancy, and may be discovered by accident, often in an insurance examination. The physician may discover it with his instrument long before the patient does with his feelings. In other cases on exertion or after a hard day a feeling of fullness comes in the crown of the head or a sense of constriction around the head, as if a tight hat were on. With the increasing sclerosis, vibrating, hissing, or singing noises may come in the ears particularly toward bed time; dizzy feelings disappearing as they pass, earlier exhaustion and a sense of increased weariness. Any or several of these symptoms may increase as arterial thickness increases and the heart hypertrophies. Shortness of breath, palpitation, a sense of fulness in the chest or neck, and additional evidences of cardiac strain may develop as the years pass. I watched the evolution of this group of symptoms in one patient over a period of nine years, and yet there were no early morning headaches, no night urine, no albumin and no casts, except rare hyaline. The functional phthalein test remained normal. Death may result from angina, cerebral hemorrhage, cardiac failure, or even uremia; here,

however, the involvement of the kidney is terminal and not initial, the renal sclerosis extra-renal in origin rather than renal as in interstitial nephritis. The following cases illustrate the evolution of the type.

Case 1. A married woman of forty-two, of nervous temperament and large capacity for worry, had borne two healthy children, managed her home, and taught elocution for twenty years. She fertilized her emotions, exaggerated her irritations, and imagined most of her difficulties. By accident her pressure was taken and found to be 200-130-70 m.m. Hg. P. 80-90. Unfortunately her medical attendant gave her the figures, and more worry. Arteries nowhere palpable. Heart normal. Twenty-four hour urine normal. No nycturia. No symptoms of renal disease. Feels well, sleeps well, plays golf. At forty-three she bore a third child, and aside from a few casts and traces of albumin in the latter months did well. After three years of observation the temporals and radials seem slightly thickened and the apex seems inclined to the left.

Case 2. A man of forty-eight years applied for insurance in December, 1915, and was refused because his pressure was 185 m.m. Hg. Four years before he was accepted, but his pressure was not taken. A slender man of 142 pounds, inclined to worry, no vacation and no exercise for sixteen years. Chews tobacco. Feet and hands cold, numb sensations in extremities, more in right leg. No night urine. B.P. 190-100-90. Pulse 72. Arteries large, palpable, tortuous. Vessels first space, 3.5 cm. wide, apex fifth space, in nipple line, heaving precordium, occasional sharp precordial pain. Urine negative except for a few hyaline casts. Phthalein test 42 per cent. Wassermann reaction negative. Improvement under rest and vacation.

Here the symptoms seem more referred to the heart. The aorta is probably dilated and the heart hypertrophied. The kidneys are doing their work. The patient thought he was well until his life insurance examination. Hypertension had probably existed for many years and preceded the sclerosis. Cardiac hypertrophy is progressive, and the renal findings are minor. His life was temperate. The absence of vacation or change is interesting.

II. Toxic Type. Arteriosclerosis is the chief lesion, and hypertension and renal disease may develop as a result of the effect of

the toxins on the kidney as well as the arteries. As Allbutt well says, this is "a convenient if somewhat heterogenous class." There are two divisions: (1) Toxic sclerosis with normal pressure and (2) toxic sclerosis with hypertension. Toxins from whatever source have affinities for three organs: (1) The heart, producing injury to the myocardium or a myocarditis. This lessens its power to hypertrophy and its ability to maintain the highest pressures or even lower levels of pressure as long as an intact myocardium. (2) The kidney, producing in the patient the symptoms of a chronic nephritis, raising pressure, and complicating a toxic sclerosis with a chronic nephritis. (3) The arteries, producing a toxic sclerosis. Oliver<sup>8</sup> found an average pressure of 122 m.m. Hg. in 84 lead workers, yet in another with symptoms of granular kidney, the pressure was 169 m.m. This last man might have had normal pressure had the lead not caused a contracted kidney. The very number of toxins and the differences in toxicity lessen our ability to note the damage done by any one, and cloud the purely arterial thickness with the renal injury. Gout certainly produces arteriosclerosis, and one may have gout, sclerosis, and normal pressure. The granular kidney may be also a result or at least an accompaniment of gout, and in addition to the first picture, we have nephritis and hypertension. Assuredly there is no always or never in medicine.

The symptoms depend upon the height of the pressure and the degree of development of the sclerosis. Premature age, cold hands and feet, lack of strength, and vague and unaccountable pains are common. This type is usually found in the course of a medical examination for some more general complaint. These patients seem forced to the shelf of life too soon. The ability of toxins to produce sclerosis is a fact firmly fixed in the medical mind of today, but it is difficult to measure the damage wrought by any one factor or toxin, much less many deleterious substances acting for years in one patient. Infectious diseases with syphilis in the lead and poisons originating outside and inside the body form the chief groups. Syphilis has probably been too often overlooked and underestimated, and intestinal auto-intoxication too much emphasized. Norris<sup>7</sup> rightly says "the literature on the latter subject is enormous and concerns itself chiefly with nebulous hypothecation." The two

following cases illustrate the multiplicity of factors classed as toxins and their result on the arteries.

Case 3. A man 56 years old entered the hospital on February 5, 1916, suffering with acute coryza and bronchitis. He appeared in good condition and in a week was as well as usual. He had syphilis at 24; acute rheumatic fever at 34, sick 9 months; had been a heavy eater, drinker, and smoker all his life, and done hard physical work as fireman and mechanic. All superficial arteries large and leathery. B. P. 110-50-60 m.m. Apex just outside mid-clavicular line. Systolic bruit over aortic area. Nycturia 1 to 3 for five years. Urine 1,014, trace albumin, few hyaline casts. Hemoglobin 75 per cent. Wassermann reaction negative.

Case 4. A switchman of 48 years was examined on March 4, 1916. He had recovered from a double tertian malaria. He had chewed tobacco for thirty years, drunk whiskey to excess all this time; had small-pox at 29, gonorrhea and malaria at intervals for three years. Heart normal. Arteries large, thickened, and tortuous. Retinal arteries normal. P. 80. B.P. 120-70-50. Wassermann reaction negative.

The interesting fact in these cases is the rather low pressure, particularly in the first case. It is probable in such cases that the myocardium and the kidney have suffered deterioration. Had hypertension existed, the heart would have been far more involved. An interesting question is why hypertension is absent. As Allbutt intimates more and more cases will probably be drawn into the toxic group as our knowledge advances. Hyperpiesia itself may be the result of toxins. Our present knowledge of the influence of the ductless glands on the arteries and pressure is far from satisfactory.

III. Renal Type. The arteriosclerosis is renal in origin and the symptoms are those of the old chronic interstitial nephritis, vascular nephritis is a newer term, the contracted granular kidney of the English. This type is really a vascular degeneration of the kidney, associated with a progressive and synchronous arteriosclerosis and hypertension. The contraction of the kidney, the rise of the pressure, and the thickening of the arteries are usually parallel processes. This speaks for the usual cases, though Gull and Sutton<sup>a</sup> record a case of contracted kidney in a youth with normal arteries and heart, and pressure unknown. Hypertension and hypertrophy of the heart seem to develop together.

High tension, particularly the diastolic, and that of 120 m.m. and more, is good reason to suspect the kidney. Added to this, early morning headaches, backaches, rapid pulse, night urine, early exhaustion, dyspnea on exertion, heavy breath, and dizziness, combined with polyuria, urine of pale color and low gravity, with albumin absent or in traces, scant hyaline and granular casts, rare red blood cells, tend to complete the picture. The following is a classic type.

Case 5. A lawyer of 45 years had been troubled for five years with increasing headaches in the early morning, backaches, weakness, irritability, and failing vision. His weight was 212, height, 5 feet, 11 inches. Normal weight for height is 170 pounds. For twenty years he had smoked constantly, overeaten, taken no exercise, worried, worked, made money and grown flabby. Face swarthy, eyes puffy and slightly exophthalmic. On November 21, 1916, he was found with a renal retinitis, B.P. 200-130-70. P. 90-100. Apex in nipple line, liver two fingers down. Aortic second accented and split. Nycturia 2 to 4 times. Urine: 1,800 c.c., 1,012, clear, trace of albumin, a few hyaline and granular casts. Small amount of urates. Functional test, 60 per cent. Arteries were thickened and sclerotic, inclined to tortuosity. Wassermann reaction negative. His eyes and his headaches sent him to his physician. Under appropriate treatment and rest improvement was marked. This patient changed his entire scheme of life and is now able to play golf. His headaches recur after heavy evening meals.

Headaches may occur in hyperpiesia and in toxic sclerosis. They do not present the early morning regularity of this type with the associated backache, though the latter is rarer than the former. Clinically one cannot distinguish between the feel of the arteries in a case of renal disease and the two previous types. The pulse in the renal group seems often of harder tension, but the superficial arteries may not appear so sclerosed as in the well developed toxic type or even hyperpiesia in the sixth or seventh decade. Indeed I have felt that the inclination to tortuosity and loops was more pronounced in the toxic and decrescent groups with normal or low pressures. Whether this tortuous and looping tendency of the artery, resulting of course from the lengthening of the vessel, has an influence in maintaining lower pressure than otherwise, I do

not know. The large, leathery type of vessels seems more characteristic of the toxic group, particularly in syphilitics. In a negro of only 36 years, I have seen the vessels of this type, signs of an aortitis, normal pressure, and an untreated syphilis of 6 years standing. The small, hard, fibrous vessel, the old whip cord metaphor, is more characteristic of the decrescent type. It requires more than sclerosis or hypertension or a few hyaline casts to make a diagnosis of the renal type, and too often on these three findings the medical man is inclined to this diagnosis, whereas only one of the other and less serious forms of arterial disease is present.

IV. Decrescent Type. An arteriosclerosis with low or normal pressure or rise proportionate to age, heart of relatively normal size, and without definite symptoms of renal disease. The arteries are more tortuous than in the other types, often beaded by calcified nodules which may be palpated by running the finger gently lengthwise the vessel, particularly if age has decreased the subcutaneous fat. The aortic second is usually greater than the pulmonic closure, but not distinctly accented. The cardiac sounds are remarkably clear. The kidney is the senile kidney, small, softer and smoother than the arteriosclerotic kidney of the first two groups, and far less granular than the interstitial kidney, with more pelvic fat, and of "little clinical importance save as a general closing in of capacity and reserve" (Allbutt). There is less nycturia than in the renal group, albumin absent or in traces, and usually rare hyaline casts. Cylindroids rare.

This is the group of age and the chimney corner. Its fixation begins in the fifties, and extends to death. The symptoms in the early and moderately developed periods are either absent or less in number and severity than in the other groups, due probably to the fact that hypertension is absent, the heart good, and the kidney function sufficient. The circulation is compatible with old age even though the vessels be sclerotic and calcified. The arterial symptoms are the symptoms of old age rather than of a type of sclerosis. The thickened arteries and fair kidneys with a good heart tell the story. As age advances the terminal stage comes through arterial, cardiac, or renal failure. It is one thing for an organ to be equal to the curtailed routine of a meagre physical life, but quite another pathology for such organs to fail completely. Here occurs the

cerebral sclerosis of the aged, often with mental failure, lapses of memory, and disturbance of nutrition. Emaciation, probably central in origin, similar to though slower than the rapid emaciation of meningitis, may proceed slowly to death. Periods of helplessness develop, involuntary movements, unconsciousness, and loss of reflexes.

Case 6. A woman of 82 years suffered occasional attacks of loss of memory, and mental inertia. Arteries round, hard and smooth, pulse 72, pressure 140-100-40 m.m. Heart normal in size and sound, aortic second closure greater than the pulmonic. Urine contained rare hyaline casts.

Case 7. A well preserved clergyman of 75, weight 160, suffered with a large prostate. His palpable arteries were all thickened and tortuous. Heart normal. Pressure 130-70-60 m.m. P. 50 to 60. Only one cast on repeated examinations of the urine.

Case 8. A tall, lean man of 78 years, erect and of good carriage, had for months lapses of memory and periods of great irritability. On March 1, 1916, he engaged in a fight with a fellow boarder about a trifle, lapsed into unconsciousness afterward, and was brought into the hospital in a dazed state. In a few hours he seemed normal mentally, and after a night's rest felt well, though he did not remember the fight or coming to the hospital. All superficial arteries small and markedly sclerotic and tortuous. Heart normal in size and position, though a slight mitral systolic murmur is heard at apex. Pulse 70, full volume. Blood pressure 150-95-55. Coarse tremor of tongue. Arcus senilis marked. Retina shows beaded arteries of cork screw type. Aortic and pulmonic sounds clear. Hemoglobin 85 per cent. Urine 24 hours' amount 960 c.c. Albumin a trace, and a few hyaline and granular casts, with mucus and cylindroids. Functional test, color appeared in 18 minutes. Temperature 97.4 deg. Fahr. Respiration 20. Wassermann reaction negative.

Case. 9. A withered, emaciated, old school teacher of 68 was seen on March 10, 1916. He lay silent in bed, slept most of the time, or lay with his eyes shut, and mumbled to himself. His mind, vacant at one hour, would at another seem normal. He had been losing in strength and mind for two years. Two months ago he had a severe cold. A month ago he lay unconscious for four days,



and since that time his urine and stools have gradually become involuntary. An active nephritis has been evident since the cold. He complains of wandering pains in joints, with alternate diarrhea and constipation. Tongue coated, breath bad, a little old man of skin and bones. Frontal and occipital headaches have increased. Arteries hard, small and tortuous, P. full 69, frequent premature contractures, and a month before on one visit his physician found it 36, probably a heart block. T. 94 to 97, only 98.2 by rectum. Apex fifth space, normal area. C-H angle acute, soft mitral systolic murmur, no edema. Lungs slightly emphysematous. Liver two fingers down, tender. B.P. 150-80-70. A<sup>2</sup> and P<sup>2</sup> faint. For a year he had a rather spastic walk, and stumbled frequently. Now K.J. decreased, ankles spastic, double Babinski and Oppenheim on left foot. Many nevi.

The cerebral arteries are probably undergoing obliterative processes in both these cases. In the last case areas of cerebral softening have no doubt developed. Case 8 shows a nephritis, but here the senile kidney is simply contracting to exhaustion. In Case 9 an acute nephritis has been imposed on a senile kidney.<sup>9</sup> The end of these decreascent cases is often days or weeks of unconsciousness, simply lying quietly and breathing life away, mumbling incoherently or in an active muttering delirium,—an atherosclerotic dementia. The clear uremia of the other groups is usually absent.

The difficulty of classification in all cases either of hypertension or sclerosis or both is admitted, but it is certainly not enough to say that a patient is arteriosclerotic or has high blood pressure. The prognosis depends not only upon the individual patient, but also upon the type of arterial disease that has fastened itself upon him. I have found the above classification applicable in sorting these cases. It is really more than an academic classification, for it is a real summary of the different groups and the cases fit into them.

After forty the circulation is the largest gate to death. Arterial failure and cerebral hemorrhage, renal failure and uremia, and cardiac failure with edema or anginal states are the three great paths to the end. Cerebral hemorrhage may occur in the toxic or decreascent groups with normal pressure just as it does in hyperpiesia or the renal group with high pressure. In one case of decreascent sclerosis there were three cerebral hemorrhages at inter-

vals. The patient is still living, though the pressure has gradually risen to 150 m.m. I have found angina more common in the hypertensive cases, though it does occur with normal pressures. Stone<sup>10</sup> has attempted to classify these cases on the basis of termination with some relation to his pressure ratio. It would seem well, however, to follow the more natural classification of Allbutt, and not wait for the terminal period to determine the group. Arteriosclerosis without hypertension is of more favorable future than with hypertension. If hypertension be permanent, cardiac hypertrophy and renal sclerosis will follow. The renal group is by far the most dangerous.

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## THE DIAGNOSIS OF PELLAGRA

By EDWARD J. WOOD

Physician to the James Walker Memorial Hospital  
Wilmington, North Carolina

Pellagra, in the Southern States, probably affects the two races about equally. It is a difficult matter to settle this point for obvious reasons. Some think that the negro is less frequently affected and a basis for this opinion is the absence in this race of uncinariasis which is one of the most striking predisposing factors in pellagra. In the present series of 100 cases there were only seven negroes, due to the fact that these cases were selected from my private practice and not from the wards.

The following table shows the distribution of the disease in age. It is arranged in decades:

<i>Decade.</i>	<i>Number of Cases.</i>
First .....	7
Second .....	11
Third .....	24
Third .....	28
Fourth .....	28
Fifth .....	17
Sixth .....	7
Seventh .....	4
Eighth .....	2

My youngest case was 22 months and oldest was 78 years. In the French literature there are reports of children as young as 3 months. The occurrence of pellagra in children under 3 years is exceptional.

In no disease are the symptoms so definite as in pellagra, and yet mistakes in diagnosis are numerous. The disease is invariably clear cut in its manifestations, and if the doubtful cases are followed for a few years, it will be found that the diagnosis was not borne out. In my own doubtful cases I have invariably found that the condition was later found to be another disease, as tropical sprue.

For diagnosis the disease picture should be separated into the skin group, which is most essential; the intestinal tract group, which is next in order of importance; the mouth group, which is least in importance, but always helpful and often present before the skin manifestations.

After 12 years' experience in a large number of cases I feel safe in the conclusion that a diagnosis is never justified in the absence of skin symptoms. This conclusion is at variance with some teachings and will cause the failure to diagnose early a few cases, but it has proven better to make this error rather than to have a larger number of errors from the other course. It is impossible for me to diagnose pellagra without the skin symptoms or a very definite history of a past occurrence.

Of the three groups of symptoms it is an almost invariable rule that two, at least, of the groups will occur. The one exception is

in children. In this class the disease is usually trivial, and it is frequently the case that extensive and perfectly characteristic skin lesions will occur and remain throughout the only symptoms.

The hundred cases on which this report is based were selected at random from private practice. The hospital cases make up another group. Of this number it is needless to repeat that the skin was affected in every instance, for the diagnosis would not have been accepted otherwise.

In this series 77 cases presented some diarrheal disturbance; 8 were habitually constipated; 15 showed no abnormality. In many of the cases the looseness was only trivial and only by close inquiry was its occurrence elicited. Frequently the patient would acknowledge that in the spring or early summer the first of the new fruit and vegetables caused a transient diarrhea. On the other hand, in most cases the diarrhea was severe and very resistant to all former treatments. In our experience this diarrhea, like the other symptoms, gives away promptly to high vitamin feeding. Nothing so clearly indicates the subsidence of an outbreak as does the cessation of this symptom, and until this occurs the gravity of the case is still a consideration. In many of the fatal cases the diarrhea has persisted long after the last remnant of skin lesion has disappeared. I have no knowledge of a fatal case of pellagra without this diarrhea. When the diarrhea is absent the case is usually of the very mild type, though these patients do not always escape the pellagra psychosis.

It should be noted in passing that pellagra has lost much of the virulence which characterized its appearance in the South during the first two years. In this period the cases were acute and fulminating, running a course of about six weeks, or even less. The death rate in my own experience was above 80 per cent. Year by year this state of things has improved, until today American pellagra is fast approaching the Italian type. From 1905 to 1908 there were many difficulties in the way of arriving at a diagnosis, because acute pellagra (except, of course, acute exacerbations) is unknown in European literature.

The mouth symptoms were present in 73 cases, and absent in 27. In many instances I have found redness of the tongue and mouth generally when the patient made no complaint. The duration of the

mouth symptoms may be so short that a failure to record them must be very frequent. The mouth in pellagra is very variously described. Experience teaches that it is far better to avoid close differentiation. To say that there is an acute stomatitis and frequently an esophagitis is enough. The tongue is uniformly red and moist. The color is deeper than in scarlet fever or in tropical sprue. It is protruded with difficulty, owing to pain. The so-called "strawberry" appearance is lacking. J. W. Babcock has noted that the redness over the hard palate about coincides with the area occupied by the plate of false teeth, and is just as definitely demarcated as the skin is. Salivation is a marked symptom in many cases, and it has seemed to me to be an indicator of the severe type of the disease.

In my own experience the mouth symptoms of sprue cause the greatest confusion with pellagra. When the other parts of the disease picture are considered, there is no occasion for any difficulty. It must be remembered that in sprue there is diarrhea and stomatitis with marked glossitis. The type of diarrhea in the two diseases is marked and will be referred to later. The skin lesions in sprue never occur, and this in itself almost justifies our position that pellagra must not be diagnosed unless this group is present. In sprue the tongue is larger, paler, and is usually furrowed. Thin says that salivation does not occur in sprue. It is an important fact that tropical sprue is occurring in appreciable numbers in the South at this time. As a rule it is rather a milder type than that recorded by British writers, whose experience is usually with the Indian type. In this American sprue the mouth symptoms are not severe. It cannot be too often reiterated that in sprue there is a high degree of secondary anemia, which does not occur in uncomplicated pellagra.

The most interesting, and naturally the most important, group of symptoms in pellagra is the skin. As before stated, no case is accepted in this group as pellagra in which skin symptoms are not present, or had not occurred. Many doubtful cases have later proven to be amebiasis or sprue.

Of this series in 97 the hands or forearms, or both, were affected. The backs of the hands and the lower portion of the posterior surfaces of the forearms are to be expected as the seat of

the pellagrous skin lesion with almost as much predilection as the throat is the seat of a diphtheritic pseudo-membrane. In a large proportion of this series there were additional locations, but the emphasis must be laid on pellagrous hands. In 39 cases the hands alone were affected. In 10 the hands and forearms, in 5 the forearms alone. In 2 cases the face and neck were the only seats, and in one case the tops of the feet. In 16 cases the feet in combination with other parts, were affected. In 8 cases the face was involved. In 6 cases covered portions of the body were affected. One of the striking features of pellagra is that the skin lesions almost invariably occur on those portions exposed to direct light: the uncovered portions. The exceptions to this are extremely interesting. Goldberger's experimentally produced cases (U. S. Public Health Reports, 1916) showed that the scrotum was the seat of the lesion in some cases. Some writers have seen fit to question Goldberger's cases because of this unusual location. In one of this series the scrotal lesion was the first to appear, but subsequent areas involved made the diagnosis of pellagra certain. In a case now under observation and not included in this series there is a definite perianal lesion, and in still another case of another series there was a perianal lesion without other skin manifestations. In this series there were other interesting departures of this sort. In one case a woman presented a skin lesion which practically covered the whole of her trunk, leaving sound skin only over the shoulders where the shoulder straps of the chemise had offered greater protection. One boy had two areas beneath the scapulae which were triangular in shape and perfectly symmetrical in every detail. Vaginitis occurs frequently. The labia majora are involved and the lesion partakes in many respects of an erythema with the same type of mucous membrane involvement as seen in the mouth.

No reference to the skin lesions of pellagra can be complete without emphasis on the symmetry of the lesions. This symmetry is so certain to appear that it is often possible after the lesion appears on one side to mark out an identical area on the other side of the same shape, size and location. The skin lesion will exactly coincide with the markings. This symmetry is the most important diagnostic point in the skin group of pellagra. A lesion otherwise suggestive

should not be counted pellagrous if symmetry is wanting. Added to symmetry is a pigmented line occurring just within the line of demarcation and known as the hyperkeratotic border of Merk. It persists long after the subsidence of every other sign of the skin lesion and is frequently all sufficient for a diagnosis. There occurs frequently in pellagra a peculiarity of the skin lesion which may be likened to the concentric layers of the oyster shell. The first outbreak of the skin is the largest and each subsequent outbreak is less in size and superimposed over the former, so that finally the last eruption is only a very small patch. The result is strikingly like the oyster shell and indicates an important point in the disease: the tendency for frequently repeated exacerbations at short intervals. Does not this suggest such a disease as scurvy rather than an infectious process? But such discussion is out of order in this paper.

The reflexes in pellagra are variously affected. In some instances autopsy shows definite tract degenerations (Tuczek: *Die anatom. u. patholog. Studien über die Pellagra*). In one case in a 17-year-old negress the columns of Gall and Burdach were definitely degenerated with no involvement of the posterior roots. In this series the reflexes were increased in 32; in 20 there was definite decrease; in 37 there were no abnormalities. In 11 cases the reflexes were mixed. One group would be exaggerated and another decreased or normal. Again, on one side there would be a different condition from the other.

Exaggeration of reflexes in my experience was usually a point of favorable prognostic significance, while depression of the reflexes occurred more frequently in grave cases.

Too often the psychosis is made the basis of a diagnosis of pellagra. This is never justified and almost certainly will lead to error. The psychosis of pellagra is too indefinite to be of diagnostic value. The only point of importance is to remember that the mental expression will always be one of profound depression.

Finally, it may be said that except in the South there can be no occasion for confusion of pellagra with any other condition. In the South in the presence of skin lesions the diagnosis offers no difficulty. When the diagnosis is ventured without this group the stools should be made the chief point of diagnostic attack. In many cases of amebiasis there occurs stomatitis and the stools

should be searched with unusual care for amebae. Sprue has been mentioned. It is destined to soon play a large part in Southern medicine. The disease is characterized by stomatitis mentioned above; by a looseness of the bowels, occurring chiefly in the morning hours, unattended with tenesmus, with large, soft, "cow-pad" stools which are acid and full of fat. A study of the feces (*Am. Jour. Med. Sci.*, 1915) shows that on the Schmidt test diet there occurs a tremendous loss of fat and nitrogen, which is not so in pellagra. The high degree of secondary anemia in sprue does not occur in pellagra.

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### SYMPTOMS VERSUS PHYSICAL SIGNS IN THE EARLY DIAGNOSIS OF PULMONARY TUBERCULOSIS

By PAUL H. RINGER  
Asheville, North Carolina

Despite the nation-wide crusade against tuberculosis, and despite undeniable progress in the early diagnosis of the disease, the fact remains that far too many cases remain unrecognized until they have reached a comparatively advanced stage. One reason for this is that the general practitioner is wont to wait until there are indubitable signs of trouble to be found on physical examination of the lungs. Now, the general practitioner, as a rule, does not see enough of the earliest physical pulmonary changes to cause him to appreciate their importance. Accurate physical diagnosis takes practice, and continued practice; and this, the busy doctor, with his multiplicity of ailments to treat, frequently does not attain. The finer changes in the lungs are not difficult to appreciate; it is not that the expert is in any way above the general practitioner, but the detection of these more minute deviations from the normal demands continued, careful time-consuming inspection, mensuration, palpation, percussion, auscultation. This the expert must use if he is to deserve the name, and this the general practitioner has not the time to do.

Many physicians are loath to make a diagnosis of pulmonary tuberculosis in the absence of tubercle bacilli from the sputum. The diagnosis can and should be suspected, if not actually made, before



there is any sputum to examine. In only  $33\frac{1}{3}$  per cent. of the early cases (not really incipient, but very slight) are bacilli to be found. What of the other  $66\frac{2}{3}$  per cent.? Must they wait before the diagnosis is made until ulceration has occurred, and a mode of exit for the bacilli has been provided? One negative examination of sputum means nothing more than that bacilli, if present, are certainly in small numbers, and no examination of sputum, whether positive or negative, is of any value unless the skill of the examiner is beyond question.

Today, as twenty years ago, the cry is for early diagnosis, and today, as twenty years ago, no doctor, no climate and no mode of treatment can do much, save in very exceptional instances, for the far-advanced case. Now the early diagnosis of pulmonary tuberculosis is no extremely difficult matter, or, to state it differently perhaps, the suspicion of the presence of pulmonary tuberculosis is one that the general practitioner can entertain as soon as the expert. For the suspicion of pulmonary tuberculosis rests on certain subjective and objective symptoms, stated by, or observed in the patient, far more than upon certain changes found in the lungs. As a matter of fact, in the diagnosis of a really early case of tuberculosis, the expert relies far more upon symptoms, in which there is little likelihood of his being mistaken or misled, than upon slight physical signs in the interpretation of which he may err. Furthermore, there should be added, that to the man dealing largely with pulmonary tuberculosis, suspicious, or slightly pathological lung findings, in the absence of symptoms, signify little more than that the patient should be observed and examined from time to time, while those same lung signs in the presence of symptoms become most convincingly diagnostic.

Physical signs are due to modifications of physical conditions in the lungs, and do not in themselves make a diagnosis. Symptoms are the evidences of the systemic effects of disease, and point the finger both to the virulence of the infecting agent and to the resisting power of the infected individual. It is a fact known to all, that the tuberculous individual, with many signs and few symptoms, gives a far better prognosis than the tuberculous individual with few signs and many symptoms.

Any three of the following six symptoms occurring in a patient

should warrant the bearing in mind the possibility of tuberculosis till its existence can be positively disproved.

1. **FATIGUE**, especially unreasonable fatigue, coming on in the course of ordinary work, and from which the patient does not quickly recover. An important point is that the patient gets up tired.

2. **LOSS OF WEIGHT**.—Moderate and persistent reduction in weight is important. The patient should be weighed twice weekly on the same scales, at the same hour of the day and in exactly the same clothes. Only thus can an accurate record be obtained for purposes of comparison.

3. **PERSISTENT ANOREXIA**, occurring without any discoverable gastric or intestinal lesion. At times there is a craving for sour articles of food. This type of anorexia, due to the toxins of the tubercle bacillus, is not benefited by the ordinary stomachics.

4. **SLIGHT TEMPERATURE ELEVATION**, over 99 deg. F., at any time of day, but especially in the afternoon. The temperature should be taken every two hours from eight A.M. to eight P.M., and the thermometer left in the mouth five minutes each time. A rise to 99 deg. immediately after a meal is of no significance, provided that within an hour normal limits are again reached. It must also be determined whether a brisk walk for thirty minutes or so sends the temperature up. If it does, this is of diagnostic importance. Of distinct importance, though of rarer occurrence, is the persistence of sub-normal temperature, the daily maximum being 97.6 to 98 and the minimum being 95.6 to 96.6; this type of temperature has practically the same significance as the slight febrile movement.

5. **RAPID PULSE**. The pulse must be counted when the patient is at rest. It is best to have the patient count the pulse himself at the same time that the temperature is being taken. Most patients become excited if some one else counts the pulse. Of course, the physician must be certain that the patient can count the pulse with reasonable accuracy. Any pulse rate persistently over 80, especially early in the morning before the patient is out of bed, is suspicious.

6. **NERVOUSNESS**. Many patients present signs of a mild degree of neurasthenia, due to the action of the toxin of the tubercle bacilli.

Though nervousness by itself is of no diagnostic value, in conjunction with other symptoms, it becomes of corroborative importance.

Three further symptoms have been reserved for consideration in a separate group. It was desired first of all to consider those symptoms of early pulmonary tuberculosis that would in no way attract the attention of patient or physician to the chest as the site of trouble. It is because these "non-specific" symptoms have been disregarded, or misinterpreted, that so many patients have been assured of the soundness of their lungs and have gone their way only to return later in ill health with the signs of tuberculosis only too plainly delineated. The three symptoms now to be mentioned are:

1. Cough; 2. Sputum; 3. Hemoptysis.

Cough and sputum will not be the subject of much consideration, for a diagnosis should be made before either of them becomes prominent. Many patients will at first deny having any cough whatsoever, but very careful questioning will reveal that they have a little morning cough, "just like anyone has." This is important; *the perfectly normal "anyone" has no morning cough.* It is axiomatic that any cough lasting over two weeks demands searching investigation, and that any sputum, whether accompanied by cough or not, should invariably be examined for tubercle bacilli, and, should such an examination prove negative, should be frequently re-examined until all likelihood of bacilli being present has disappeared.

Hemoptysis demands a few additional words. Unfortunately patients with a small initial hemoptysis (under one ounce) are still frequently told that "the blood came in all probability from the throat," for no physical signs can be heard in the lungs. It is difficult to censure such advice too severely. Blood may come from the throat, but when it does, it usually escapes from a varicose vein, which can be readily seen, and which often has to be tied or compressed before the hemorrhage can be stopped. Obscure pharyngeal or laryngeal hemorrhages are excessively rare. Blood that "probably came from the throat" in reality came from the lungs. Initial hemoptysis is Nature's red flag of warning to the patient, and how often is that warning unnoticed! Every case of hemoptysis must be considered as of tuberculous origin until this assumption has been wholly disproved. By adherence to this rule but few

mistakes will be made. One or two mouthfuls of blood that come up and are gone are important, and in 98 per cent. of the cases alone constitute enough evidence for the diagnosis of pulmonary tuberculosis.

If we consider for a moment the two groups of symptoms as one, mention must be made of "Norris' quartet." These are:

1. Rapid pulse,
2. Slight afternoon temperature,
3. Slight loss of weight,
4. Slight cough.

It is held by some that the presence of any two of these symptoms warrants a diagnosis of tuberculosis; the writer cannot fully agree with this statement. Of course, but few patients in the really early stage of the disease are going to present themselves, showing all the symptoms that have now been briefly considered. As mentioned, however, the presence of any three of the six composing the first group, is sufficient to warrant the strong suspicion of pulmonary tuberculosis, and when to any three of the first group is added any one of the second group, the diagnosis becomes almost beyond question, and must be persisted in until it is absolutely proven to be fallacious.

If the general practitioner suspects tuberculosis in a given case, should he make use of the various specific tests in order to reach a positive conclusion? I refer, of course, to the tuberculin tests. Broadly speaking, it is unwise for the inexperienced to work much with tuberculin, especially in the administration of the subcutaneous test, unless under the eye of one accustomed to the procedure. The dosage, the intervals between doses, the standard of what constitutes and what does not constitute a reaction, all these matters necessitate familiarity with, and frequent use of, the product. The Calmette ocular test is well left untouched, and indeed is hardly used at present. The Moro test, with tuberculin ointment, is not of much value. Almost all physicians use the von Pirquet test, which to be sure, is extremely simple and quite harmless, but the interpretation of this test is the important point. Following faultless technic, a negative reaction in any patient is of value. A positive reaction is of great value under two years of age, decreases in value with every year up to eight, and from then on is valueless,

indicating simply that the patient has been infected with tuberculosis. Many adult patients are seen who have been told that a positive von Pirquet was a very strong diagnostic point in their particular case. Thus, we see that a tentative diagnosis of early pulmonary tuberculosis depends far more upon common sense and the correct appreciation and interpretation of a few symptoms than upon extraordinary skill at percussion and auscultation, or in the reading of a radiograph. There are, of course, many conditions that are ushered in by a combination of the symptoms that have been dwelt upon; but examination of one kind or another will in most cases clear up the condition. The plea in this short article is for more frequent consideration of the possibility, nay even of the probability of tuberculosis, the most common of all infections, even in the absence of physical signs pointing focally to the seat of the lesion. We have seen that this diagnosis is one that any competent and conscientious physician should make and can make, and there follows that with early diagnosis will come proper advice, correct treatment and an increase in the percentage of recoveries.

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#### A CLINICAL CONSIDERATION OF TEMPERATURE OF SO-CALLED OBSCURE ORIGIN IN INFANTS AND CHILDREN

By H. LOWENBURG

Pediatrist to the Mt. Sinai Hospital; Pediatrist to the Jewish Hospital and  
to the Jewish Maternity Hospital, etc.  
Philadelphia

Infectious processes occurring in the human body depend for their symptomatology largely upon three factors—the character of the microorganism, the resistance of the individual and the location of the infection. All infections by the same organism are similar as to symptom-complexes, except in so far as these may be modified by the latter two factors, especially by the locality involved. It is also true that many of the general features of infection by dissimilar microorganisms are often identical, or if not so, quite often similar. Of the general features of infection only temperature or fever will be discussed in this paper. This is the characteristic most uniformly common to the majority of infections. That the nature of the temperature curve may be similar in many types of infec-

tion, is from the standpoint of diagnosis, simultaneously a detriment and an aid of much value. Fever, per se, almost uniformly presupposes an infection per se, but not uncommonly beclouds the issue as to the nature of the infection, especially when symptoms of localization are for the time being, at least, not manifest. Nowhere is this difficulty more frequently experienced than at the bedside of a sick infant or child. In some instances, after the most thorough and comprehensive search, including the employment of every available clinical and laboratory avenue of information and detection, the riddle may remain unsolved. While, as stated, fever, as encountered by the clinical pediatricist, will therefore embrace the scope of this discussion, emphasis of characteristic features occurring in the nosology of certain infections will also be made as they may appear to be of assistance in elucidating the etiologic factor responsible for the fever in an individual case.

Given an infant child with temperature, what are the diagnostic possibilities and what methods of procedure may be undertaken to establish the correct diagnosis? As to the first, it will not be necessary to enumerate the host of illnesses associated with fever to which childhood is heir. As we proceed, a certain number of those conditions commonly regarded as obscure, or, at least, often overlooked, will be considered with reference to any particular peculiarities. As to the second, it is first necessary to establish that a true infection is present or that it is not. Much may be learned from the study of the temperature chart, but in the beginning of an illness this is not always possible. Certain types of fever may indicate certain types of bacterial infection, but not the center of activity. Thus we recognize the continued fever of typhoid, the intermittent character of the streptococcic curve, the irregularity of epidemic meningitis, the critical fall of lobar pneumonia, but, at the outset, we have, by the very nature of things, no fever curve to guide us. My experience would cause me to emphasize the value and comfort afforded to the physician by a simple leukocyte count or this with a differential count at the very onset of any febrile movement in a child. It permits him, in a broad way, immediately, in most instances, to properly catalogue, as it were, any case of fever. Leukocytosis is either (a) present or (b) absent.

(a) Leukocytosis is present. A long list of possible infections immediately becomes available for consideration; at the same time a larger number of another type can be eliminated. Practically all infections associated with leukocytosis are inflammatory in nature. In this respect a differential count is of material assistance. A count of 20,000 or over would immediately put a case in the pneumonia class even in the absence of the signs of consolidation, although I will here record my opinion that the diagnosis of central pneumonia, so-called, or rather of pneumonia throughout the course of which physical signs are constantly absent, or at least undemonstrable, should be taken *cum grano salis*. In these cases the X-ray will, as a rule, consistently eliminate consolidation and the diagnosis must be sought elsewhere or else it will demonstrate the pneumonia as sub-pleural and proceeding toward the hilum. A differential count here may be misleading for polynucleosis as high as 80 per cent. is commonly present in uncomplicated lobar pneumonia. It must not be regarded as indicative of pus under these circumstances.

Leukocytosis, therefore, forestalls the opinion that an immediate subsidence of the illness under consideration will ensue, and indicates that a more or less speedy localization of the process can be expected. The detection of the seat of this, by careful physical examination and by other means, constitutes the correct diagnosis of the case.

The other acute inflammatory conditions attended by leukocytosis will be indicated as we proceed.

(b) Leukocytosis is absent. A host of acute inflammatory infectious processes are, without further investigation, immediately excluded. Thus, for example, lobar pneumonia or a pus infection of any type would scarcely be considered in an otherwise healthy child who presented a normal leukocyte count and a temperature of 104 deg. F. Among certain infectious diseases known to be associated with a normal white cell count, or even a leukopenia, may prominently be mentioned typhoid fever, malaria, a pure tuberculous infection and the later stages of influenza. I regard the association of a continuously high temperature with a normal leukocyte count or a leukopenia, as eminently suggestive of typhoid fever and have repeatedly correctly risked this diagnosis days be-

fore the appearance of the Widal reaction. Thus, too, if it be possible to exclude clinically most of the exanthemata, and with the established absence of those diseases just enumerated as known to be without leukocytosis, the association of a normal count with high fever in an otherwise healthy subject would exclude all *serious* acute infectious inflammatory and non-inflammatory processes. Thus the leukocyte count possesses a very important immediate prognostic, as well as a diagnostic significance, and should, if possible, be estimated at the outset of all febrile movements in children. Its technic is simple and should be mastered by every practitioner.

The determination, therefore, of the presence or the absence of leukocytosis permits of a clear interpretation of physical findings. In fact, it materially assists in indicating what avenues of investigation should be pursued. It also permits of the rough classification of any febrile movement into one of two classes, (a) infectious which may be either inflammatory or non-inflammatory and (b) non-infectious and of probable short duration. Even though the line may not be drawn with absolute certainty, the distinction indicated is of immense assistance in reaching a correct conclusion in an individual case. The reason for this is that in some conditions where leukocytosis is present the increase may be very slight or it may not be constant, varying as the stage of the disease, notably in influenza. Certain diseases overlooked with more or less frequency will now be briefly discussed.

*Otitis Media, Furuncle of the Aural Canal.* Strange as it may seem, the profession in general gives but perfunctory attention to aural examination in infants and children sick with fever, in spite of the emphasis which this topic has received from pediatricists and otologists alike. The head mirror and speculum are useless in their hands. To one who has witnessed the gyrations and useless gymnastics of an inexperienced physician attempting to focus reflected light into the aural canal, the sight would be ludicrous were it not so pathetic and exasperating. Unfortunately, this criticism must apply to some pediatricists as well, one leader confessing to me his inability to make this examination. No physical examination of a sick child is complete without an inspection of the ear drum and of the aural canal. The skill to do this is readily acquired by any



one willing to be instructed. No expensive apparatus is necessary. Personally, I prefer reflected light to direct illumination, and have found little satisfaction from the various lamps upon the market. They are usually out of order when most needed and very few of them permit the removal of wax which might obstruct the view while the illumination is being made. A head mirror, a speculum, a tallow candle, a slender ear applicator and a wisp of cotton with a darkened room are the only necessary essentials.

Otitis and furunculosis are always associated with leukocytosis. The count averages between 12,000 and 18,000 with an increase in the polymorphonuclears. Neither condition need necessarily be associated with much if any pain, especially otitis. This is even true of children of an age sufficient to permit them to distinguish locality. This fact requires emphasis, as there are those who still believe that pain or earache is a condition sine qua non with reference to acute inflammation of the middle ear.

*Thrombosis of the Lateral Sinus.* The temperature does not always fall to normal immediately after rupture or puncture of an inflamed drum head. The persistence of temperature is often erroneously regarded as indicative of complications. However, the likelihood of septic thrombosis of the lateral sinus becomes manifest when the temperature commences to fluctuate widely between normal and 104 deg. or 105 deg. or 106 deg. F., especially if signs of meningeal irritation appear. Leukocytosis of over 20,000 with polynucleosis obtains. A blood culture made at the height of a temperature exacerbation will usually reveal the presence of hemolytic streptococci. These cases are very difficult. Many may only be suspected and are verified only by operation. The bacteremia is not always demonstrable unless the specimen be secured at the moment when the blood stream is flooded with microorganisms, and as indicated this usually occurs when the fever is at its highest. In any event, this morbid entity must always be considered as a possibility in any case wherein fluctuating temperature persists after the discharge of pus from the ear.

*Mastoiditis.* This will rarely be overlooked if the mastoid be included in the general examination of any child, particularly if there be a history of either acute or chronic otitis media. A slight degree of mastoiditis accompanies nearly every attack of acute otitis media. Many of these cases subside without surgery.

*Cervical Adenitis* may be responsible for an apparently unexplainable temperature. When *tuberculous*, unless mixed infection be present, there is no leukocytosis. If infection be present, the count may reach 20,000 and over. The detection of adenitis, when deep-seated and without external swelling, as a cause for fever, may be difficult. However, sensitiveness may be revealed by palpation. Obscurity may persist for days. In fact, if resolution sets in early, the process may never be revealed. In other cases swelling is rapid or gradually increases and is immediately noticeable upon inspection. Localization and suppuration rapidly ensue in some cases. In others, suppuration seems stayed and a hard, tender, indurated swelling remains for weeks and then slowly subsides, or one or more chains of glands become enlarged and tender, but do not suppurate. Usually a *focal lesion* in the *mouth* or on the *tonsils*, a *decayed tooth*, *stomatitis* or *tonsillitis*, or even *tonsillar diphtheria* may be demonstrated as the cause. In all cases of fever, therefore, the superficial and deep cervical lymphatics must be carefully interrogated.

*Disease of the Scalp.* *Furunculosis*, *eczema* and *pediculi* may be a simple, apparent and yet unnoticed means of causing a febrile movement. Infection may enter through a break in continuity of the hairy scalp, hence this portion of the head should always be scrutinized.

*Rhinitis*, *Nasopharyngitis*, *Nasal Diphtheria*. A child with a running nose should be an object of suspicion, especially if he has a fever. A *simple catarrhal rhinitis* is often overlooked and regarded as of too little consequence to be responsible for a temperature of 102 deg. F. or so because it is so common. This phenomenon occurs especially in susceptible babies of nervous temperament. It may remain localized, or, more commonly, inaugurates a general catarrhal process resulting in *pharyngitis* or *bronchitis*, which prolongs the duration of the fever. All of these conditions are accompanied by mild leukocytosis. Whether the process remains localized or progresses, the greatest infection lodges at its seat of origin in the nose and naso-pharynx and the temperature may be made to subside speedily by rendering these parts aseptic or nearly so. Reference will again be made to this.

A pussy nasal discharge tinged with blood, especially if it be

unilateral and with slight fever, should immediately create the suspicion of nasal diphtheria.

*Sore Mouth.* It does not seem possible that anything so self-evident as inflammatory or ulcerative phenomena of the buccal, lingual or gingival mucosae could escape notice. Nevertheless, instances have been encountered where the condition of the mouth was ignored even when the throat had been inspected. This results from the fact that stomatitis and Vincent's disease, for instance, and similar conditions are not kept sufficiently in mind. Therefore, in any case of temperature, these parts should receive careful attention and the etiologic relationship to apparently obscure fever should be considered of any abnormality noted.

*Retropharyngeal, Peritonsillar Abscess.* Strange as it may seem, these are often ignored, especially the former. Particularly is this so before the mass reaches a size sufficient to produce symptoms of pharyngeal obstruction, the most important of which is the pathognomic sound referable to the vibration of the soft palate. Swift digital palpation of the naso-pharynx, the pharynx and the tonsils should therefore conclude the examination of every child in whom the origin of fever cannot be explained. Valuable diagnostic information is often obtained by this maneuver, which is practically painless. At first a hard, non-fluctuating mass is felt on the post-pharyngeal wall. It later enlarges and fluctuates and causes the symptoms of obstruction to which reference has just been made. The trained ear is able, by the sound alone, to distinguish this condition from *laryngeal diphtheria*, with which it is commonly confused. Laryngeal diphtheria may also cause fever. It is, however, considerably lower and may be normal. Leukocytosis with polynucleosis also distinguishes retropharyngeal abscess.

*Appendicitis, Pneumonia and Pneumococic Peritonitis.* Who, accustomed to treating children, has not stood dumb and vacillating at the bedside of a critically sick child and felt his utter helplessness and despair, meanwhile fully competent to state the classical symptoms of each one of these diseases? Pneumonia with abdominal pain! Pneumonia without physical signs! The first undoubtedly constitutes a distinct entity. Does the latter? The question is pertinent. My own opinion, the product of several sad experiences, is that the diagnosis of pneumonia, if physical signs

remain absent, should be accepted with considerable hesitancy. Continued experience convinces me of the correctness of this view. Pneumonia is a very convenient diagnosis and the temptation to make it, especially during the winter months, is very great. The facial appearance in peritonitis is characteristic and helpful, but, unfortunately, it likewise predicates a fatal outcome. A cold nose and an ashen hue about the lips and nostrils, I have too often learned, indicates a fatal peritonitis of one type or another—for instance, from a ruptured appendix or from a primary pneumococcic infection. Those are the most puzzling types, which, having a suggestive but a typical lesion in the lung, also exhibit some abdominal symptoms. It is impossible in most of the cases of this type to differentiate between lung and abdominal disease until recovery or death ensues or at the operation table. From simple appendicitis, the difficulties, while great, are not so extreme. Slight abdominal rigidity over McBurney's point is suggestive of appendicitis, especially if pressure over this region, while the child lies prone, causes the patient to consistently flex his right thigh upon his abdomen. This is often the only evidence of objective pain. Subjective pain may be entirely absent as well. Rectal palpation may reveal tenderness in the right illeocecal region. In case of great doubt the patient is best observed in a hospital. An X-ray examination of the lung may also be made. Its negative value is great in excluding *pulmonary* disease. Its positive value does not exclude *abdominal* disease even though a demonstrable pneumonia be present, as I have experienced. *Pneumococcic peritonitis* occurs, though with rarity, as a complication of pneumonia. This must be borne in mind when diagnosing abdominal distension as a simple complicating tympanitis.

Simple catarrhal appendicitis, presenting but mild symptoms, I am sure, may be responsible for an apparently obscure rise in temperature. Such a case was recently seen with Dr. Herman Kauders.

*Endocarditis* is often overlooked. I am meeting it with greater frequency as I become more circumspect and persistent with reference to cardiac examination. Obscure joint or muscle pains may be present or may have passed away. Mild catarrhal or follicular tonsillitis or mild chorea may have come and gone. The history is often very meager indeed as to furnishing a clue to the primary

source of infection. So often do I experience this that I have come to regard primary endocarditis as a distinct entity, primary in nature and responsible for a certain type of mild initially unexplained morbidity in children. They are tired, languid, anemic, run a slight temperature of  $99\frac{1}{2}$  deg. to 101 deg. F. and have a very rapid, weak pulse as the most characteristic and suggestive feature of this ensemble. If these children are examined daily sooner or later will appear a soft blowing systolic murmur, which becomes louder and harsher and permanent. There is leukocytosis.

In still other cases the child becomes acutely ill with temperature. No cause is demonstrable. The temperature subsides, but not quite to normal. The child is allowed to leave its bed, as there are no urgent symptoms. The fever, mild in type, continues noticed or unnoticed. There is a sudden relapse and the child goes back to bed or is taken to the physician, who again interrogates the heart. The diagnosis becomes clear as a soft, systolic murmur is detected. Endocarditis in this case, existed from the beginning, may even have been suspected, but unfortunately remained unrecognized until sufficient mechanical damage to give rise to the murmur ensued.

*Pericarditis* is rare and is usually overlooked. It may be either primary or secondary.

*Rheumatism, Epiphysitis, Periostitis, Osteomyelitis and Scurvy.* Any one of these may be responsible for temperature. All are associated with leukocytosis. In all save scurvy, unless this be complicated by an infection, polymorphonucleosis obtains. Hemorrhages occurring under the periosteum and elsewhere are responsible for leukocytosis in scurvy. Common as it is to confuse rheumatism and scurvy, the presence of temperature in the latter disease often adds to the confusion and may serve as an argument for the uninformed to insist upon the diagnosis of rheumatism when scurvy is present. The temperature in scurvy depends upon the absorption of the disintegrating but aseptic blood clot. The manifestations of rheumatism in children are commonly and amazingly evanescent. Deceptive joint and muscle symptoms, associated with mild fever and leukocytosis, are frequently rheumatic, although they may be entirely ignored or ascribed to growing pains or influenza. Their correct status becomes manifest only after the development of a cardiac lesion.

*Epiphysitis*, independent either of rheumatism or of scurvy, occurs in infants. Tenderness in the region of the epiphysis, especially of the radius, associated with fever is significant particularly in the breast-fed. The degree of swelling and of tenderness may be so slight as to be readily overlooked. The same symptoms occurring in the artificially fed should create the suspicion of scurvy.

*Periostitis* and *Osteomyelitis* should always be considered in the presence of fever, and all the bones, including the ribs, should be investigated for objective pain. The seats of such inflammatory processes being of necessity deep, their detection may be difficult or entirely overlooked until local redness and swelling appear, unless the bones be carefully palpated.

*Pyelitis* has received considerable emphasis from many authors as being quite a common cause of mystifying temperature, especially in female infants. My own experience does not contribute to the correctness of this view. In an extensive hospital experience, I have met with but very few instances. Urinalysis, pursued as a routine procedure, will disclose the presence of this condition. The temperature is always irregular and often decidedly intermittent.

*Chorea*. True chorea, as well as that autotoxic state, which for the want of a better term may be designated as the "*neuropathic diathesis*," are often associated with irregular excursions of temperature. The rise may be very moderate ( $99\frac{1}{2}$  deg. to 100 deg. F.), but is continuous for long periods and is increased by exercise and excitement, or there may be sharp exacerbations dependent upon slight irritations. These rises are best explained upon the basis that for some reason, usually toxic, these patients have an unduly sensitive nervous system and that the heat-regulating mechanism partakes of this nervous instability. A mild leukocytosis, toxic, no doubt, is often present. These cases are commonly suspected as being tuberculous. In fact, it is not always easy to separate them from mild, deep-seated glandular tuberculosis, except by a careful history, giving to each factor its full value in the nosology of the case, by making a tuberculosis test and a careful blood count. This confusion is particularly liable to occur in those cases where the manifestations of chorea are not frank,

but where the general aspect of the patient must be summed up under that vague but withal comprehensive term "nervous." It is necessary, however, to emphasize that nervousness per se cannot cause temperature of any duration. These cases are better understood when the so-called nervousness itself as well as the temperature are regarded as being dependent upon an underlying toxic factor, itself not visible, but its presence made known only by its manifestations—to wit, the temperature, etc. In this connection careful examination of many of these little patients will reveal a *foul-mouth*, *carious*, *decideous* and *secondary teeth*, *chronic running noses* dependent upon *sinus disease*, *adenoids*, *caseous tonsils*, *constipation* and *worms*. Many of these children have enuresis and night terrors. Any one of the foregoing conditions may be responsible for the febrile movement and the nervousness, and even for choreic manifestations, and in no instance will recovery ensue unless the offending etiologic factor be removed. A recital of these various conditions is sufficient, without further discussion to direct the physicians' thoughts and acts into the proper channels.

*Dentition.* Is dentition, admittedly a physiologic process, per se, ever responsible for high temperature in infants? This topic has been much discussed by eminent authority. Unfortunately those who maintain that temperature never depends upon teething are forced to this position by the recklessness with which the average practitioner is ready to describe this as the causative agent in nearly every case of fever which he is unable to explain, providing it occurs during the teething period. Briefly, it may be stated that in the nervous infant the actual passage of the tooth through the gum may be responsible for irritability and temperature of short duration. Briefly, likewise, the following dictum must be established, viz., that it is necessary to exhaust the diagnostic possibilities of every case before the rise in temperature be ascribed to dentition. Indirectly dentition by diminishing the food tolerance may be responsible for digestive and febrile disturbances, if the quantity and quality of the food be not reduced. This is an important practical consideration.

*Influenza and Bronchitis.* A simple bronchitis may be overlooked as a cause for temperature. Influenza, on the other hand, is

a much abused disease and must often bear the brunt of the physician's inability to diagnose his case with certainty. It bears the same relation to acute febrile diseases as neurasthenia does to chronic afebrile conditions. It is a dust heap for ignorance! Unfortunately, we, at present, possess no easy and reliable means of recognizing this affection by laboratory means, and unfortunately likewise the disease is protean in its manifestations. Under these circumstances true influenza can never be diagnosed with certainty unless there be present a large number of similar cases in a community. Under other circumstances the correct diagnosis may only be reached by exclusion. The leukocyte count shows a slight increase in the beginning, but speedily falls to normal, i. e., days before the temperature touches this point.

*Empyema.* It requires no argument to establish the fact that fever may be due to empyema, and yet how many of these cases does the consultant meet which have been overlooked? In empyema of long standing the temperature may be but slightly elevated, normal, or even sub-normal, and there may be no leukocyte process. The reason so many of these cases are overlooked is because the physical signs, as they occur in children or rather infants, are not well understood. For instance, it is very common indeed, where the chest is full of pus, to note practically no interference with the transmission of the breath sounds which are often distinctly tubular. An extensive area of impaired resonance or even of flatness, out of proportion to the degree of dyspnea, which is often very slight or absent, is the most suggestive and reliable diagnostic feature of these cases. In other words, if the extensive area of impairment depended upon lung consolidation the degree of dyspnea would be great. By massive percussion or slapping with the flat of the hand the degree and the extensiveness of the impairment, as well as the sense of resistance, are better appreciated than by finger percussion. Likewise, impairment in either axilla is always suggestive of fluid. Change of dullness, with change of position has been of very little service to me in dealing with infants particularly.

*Poliomyelitis and Meningitides.* In a brief space it is impossible to indicate the protean manifestations of these morbid entities. The temperature in the former is usually high at the onset, but



irregular in its course. All unexplained temperatures occurring in summer-time, in particular, especially if an epidemic be raging, should lead to lumbar puncture and a cell count of the cerebrospinal fluid. In the beginning the cells are high, with a preponderance of lymphocytes, and the globulin is low. As the case progresses these conditions are reversed. The normal cell count is from 7 to 8 to the c.m. The fluid is more or less clear and closely resembles that of tuberculosis meningitis. Of course the history is different in the latter disease; there is no epidemic; a film forms in the fluid and the tubercle bacillus is demonstrable.

Epidemic cerebrospinal meningitis has no stereotyped method of onset. Consciousness may be lost at once. It may not be lost at all. Convulsions may be present, but they are more often absent in some epidemics. Cervical rigidity may be mild or severe. Kernig's, Babinski's and Brudzinski's signs may all be present in a single case or may all be absent, or only one of them may be present. The fever may be very high or very low, or absent, or even subnormal in the rectum, as I have seen in a fatal case. In any event, it is always irregular and is absolutely valueless as a diagnostic criterion. The association of intense, severe, cracking headache, propulsive vomiting and irregular pulse, usually slowed, is the important point of departure in reaching a correct conclusion and should immediately lead to lumbar puncture, which reveals a more or less cloudy fluid containing intra-cellular and extra-cellular Gram negative meningococci.

The other types of meningitis—except pneumococcic and influenza, are usually secondary to a focus of infection, notably the ear or mastoid. In any event, the microscope must be the court of last resort in determining the type of infection. In all these varieties the temperature, while irregular, is liable to be higher.

*Typhoid Fever, Malaria and the Various Manifestations of Tuberculosis* must not be overlooked in any case of apparently obscure fever which lasts any length of time. The association of a leukopenia and high fever has already been referred to as being extremely suggestive of typhoid fever. The Widal reaction must also be rigidly and persistently sought.

Malaria must be determined by the microscope. General miliary tuberculosis and tuberculous peritonitis are associated with irregu-

lar forms of fever and must always be considered in the presence of leukopenia, weakness, anemia, obscure digestive phenomena, especially colicky pains occurring in older children and tenderness over the liver. Glandular and pulmonary tuberculosis are sufficiently common to require no further emphasis.

*Syphilis.* I have met on two occasions, and reported many years ago, and others have done likewise, obscure, unexplainable temperature which yielded to massive doses of potassium iodid and mercury. To-day, should the suspicion of syphilis arise in this connection, the Wassermann test will probably decide the issue, although where the history offers the least suspicion, even with the absence of a positive Wassermann, I should advise the use of ascending dose of potassium iodid.

*Obscure Furunculosis*, especially of the scalp, may cause temperature. Therefore this part of the anatomy, especially if there be present eczema or crust formations, should always be inspected. Infected eczema or burns must likewise not be overlooked. The fact in itself that they are so manifest may cause them to be disregarded as the cause for a rise in temperature.

*Erysipelas.* The necessity of stripping every child completely at each visit and of inspecting every part of its body most carefully until the diagnosis is established is emphasized by the fact that the smallest spot of beginning erysipelas, especially about the muco-cutaneous junction of the vulva of infants, may be responsible for a temperature of 104 to 106 deg. F.

*Hepatic Disease*, especially catarrhal jaundice, usually has a very mild temperature in the beginning. It is soon lost and gives place to a subnormal temperature and a slow pulse. It occurs occasionally in children and should be borne in mind.

The *Acute Infectious Diseases* need hardly be mentioned as causes for obscure fever. However, the issue is often in doubt until the characteristic rashes appear, unless the premonitory features are well known and sought. In the majority of instances, however, it is necessary to await the appearance of the exanthem.

*New-born.* With reference to their etiology, temperature in the new-born presents an intensely interesting but practically an untilled field. The various manifestations of *sepsis neonatorum* are the most common, but even these do not always appear easy of detection.

Many other conditions of infancy and childhood cause temperature, some obscure in nature and some less so. The more important ones have been touched upon and the proper methods of investigation have been indicated. In any event success will only attend a thorough study of the individual case in which the diagnostic possibilities have been exhausted.

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## GASTRIC DISTURBANCE IN GALL-BLADDER DISEASE

By WILLIAM FITCH CHENEY

Clinical Professor of Medicine, Stanford University Medical School  
San Francisco, California

In sifting the meaning of "stomach trouble," three possibilities always present themselves; *first*, the symptoms may be due to organic disease of the stomach itself; *second*, they may be due entirely to disease elsewhere, without pathologic change of any kind in the stomach; *third*, they may be due to neurasthenia and cannot be traced to any demonstrable change in stomach or any other organ. The first group naturally comes to mind at once when the patient's complaint is of gastric disturbance; and cannot be eliminated until careful investigation of all sorts has excluded it. The second group, however, is a large one, and is now recognized as including many cases formerly thought to belong in the first. The third group is a steadily diminishing one, as experience grows, and no case should ever be explained in this way until all evidence that may supply a different interpretation has been sought for and considered.

In the second group the two great factors in producing gastric disturbance are chronic appendicitis and chronic cholecystitis; there are numerous others, but these two stand out conspicuously. Each plays an important part; but both are alike in this, that no medical or dietetic treatment directed to the stomach does any permanent good until the underlying pathology elsewhere is recognized and eliminated. At this time the questions chosen for consideration are the influence of gall-bladder disease in producing gastric disturbance and how to recognize "stomach trouble" really due to chronic cholecystitis and cholelithiasis.

This is no new theme. The relation between the two has long been known and described. But the writer, by reviewing certain

cases diagnosed as disease of gall-bladder, has endeavored to investigate the frequency and character of gastric manifestations encountered among these. During the five years, from January 1, 1912, to January 1, 1917, there have been 62 cases assigned to this diagnostic group; and as there have been 2,116 histories recorded during this time, all of them problems in internal medicine only, the first lesson learned is that gall-bladder disease does not form a large proportion of such material. Among the 62 cases, 19 have been looked upon as chronic cholecystitis and 43 as cholelithiasis. Classifying these with reference to gastric disturbance, they can be subdivided into three groups: 1. Those without any complaint of stomach trouble between the characteristic attacks of gall-bladder disease. 2. Those with mixed picture, of gall-bladder attacks at longer or shorter intervals and more or less constant stomach trouble between times. 3. Those with gastric disturbance predominant and gall-bladder attacks so slight, so infrequent or so conspicuous by their absence that no proof exists for a long period that the gall-bladder is the real source of the ailment.

1. In the first group there have been 30 cases; with definite, characteristic attacks of biliary colic, but no complaint of indigestion or of food disagreeing or of any gastric disturbance between the attacks. Of these cases five have been diagnosed cholecystitis; and four of these have been operated upon and the pathology proven, with negative findings on direct examination of stomach. The other 25 have been diagnosed cholelithiasis; and of these 11 have been operated upon and the gall-stones found, but no pathology in the stomach. It seems reasonably clear, therefore, that when no chronic gastric disturbance exists and the history clearly points to the gall-bladder as the seat of disease, we are not likely to err in diagnosis between the two. As regards gastric analysis, it was not made as a routine in all these cases where there was no complaint of stomach, but was made in 13. Of these, five showed hyperacidity, with total acidity above 65; six showed subacidity, with total acidity under 30, and two gave a normal total acidity, one 50 and one 56. The question arises why, with decided abnormality of secretion, no symptoms of gastric disturbance were present in 11 of these cases.

2. In the second group there were 28 cases; with constant com-

plaint of distress produced by food as well as intercurrent attacks of severe pain in the right hypochondrium, corresponding in description to biliary colic. These patients do not all have the same story to tell about their stomach trouble by any means, and there is no gastric history diagnostic of gall-bladder disease. Some complain of loss of appetite, distress soon after eating, a feeling of fullness, bloating and distension, much belching of gas, constant nausea and frequent vomiting; others make "sour stomach" the burden of their story, acidity, burning, sour eructations, and pain; while still others complain of consciousness of their stomach at all times, no matter what they eat. The danger in these cases is that the gastric history will have so impressed the patient that the attacks of biliary colic are forgotten or suppressed in the relation of symptoms. These attacks usually come at long intervals, while the digestive disturbance is constant. Four cases of this group diagnosed as chronic cholecystitis, were operated upon, and the gastric symptoms disappeared after drainage of gall-bladder. Three cases diagnosed cholelithiasis and operated upon likewise lost their stomach trouble after the removal of gallstones and drainage of gall-bladder. Of the remaining 21, not operated upon but treated medically, many were improved, but none permanently cured. As regards gastric analysis, the most common finding with this group was hyperacidity; 15 of the 27 showed this condition, but 7 showed a decided subacidity and 5 gave an analysis within normal limits. Therefore, as with stomach history, so with gastric analysis, we must conclude that there is no finding we may always expect to meet in association with gall-bladder disease. Just as there may be no stomach trouble whatever in the interval between attacks of biliary colic, or symptoms corresponding to gastric ulcer or chronic gastritis or gastric neurosis; so there may be found hyperacidity, subacidity, anacidity or normal gastric juice.

3. The most puzzling cases are those of persistent stomach trouble of one kind or another, really due to gall-bladder disease, where no history can be elicited that would suggest the gall-bladder as the organ at fault. There have been four such recorded in this group, and they all gave rise to so much difficulty in diagnosis that a detailed account of each is justified. *First*, a man aged 53, seen first in February, 1912, complained of ailing for three months with com-

plete loss of appetite, a feeling of fullness and oppression soon after eating, much belching and a loss of weight of 20 pounds. He never had any pain in his stomach or gall-bladder area. Physical examination was entirely negative as regards stomach and right upper quadrant. Analysis after a test meal showed complete achylia, the total acidity being 2 and no free HCl present by any test, with much thick, ropy mucus. The clinical diagnosis was chronic gastritis; but as no treatment improved his condition and he continued to lose in weight steadily, exploratory operation was done for possible malignancy. No disease of stomach was found but a large single gall-stone filling the gall-bladder, measuring after removal 3 by 2 c.m. Following this operation the patient's symptoms all disappeared.

*Second*, a woman aged 52, seen in June, 1913, had been ailing since the preceding Christmas; with complaint that her food seemed to stop on the way down and cause great pain; sometimes she could get it up again, sometimes, after much suffering, it ultimately passed on. At first this condition was not constant, but gradually had become so; at first only solids caused trouble, but at last practically all food and drink came up soon after they were swallowed, even before she finished her meal. She had no pain except when she tried to eat, but had lost 20 pounds in weight and grown very weak because she could keep so little nourishment. Physical examination was negative over chest and abdomen. After a test meal, on the first attempt the stomach tube was arrested 35 c.m. from incisor teeth, and could not be passed further; but the next day it did enter the stomach, and the contents so obtained gave a total acidity of 64 with free HCl 44. Radiographic plates showed an obstruction at the cardiac orifice and a dilated oesophagus above. The diagnosis was clearly cardiospasm, probably due to gastric ulcer at the cardiac end. At operation, however, no organic disease of any kind was found in the stomach; but two large stones in the gall-bladder, mulberry in character. After their removal and gall-bladder drainage, the cardiospasm never returned.

*Third*, a man aged 50, first seen in September, 1908, had been suffering from "sour stomach" for at least ten years, better and worse in spells, but never entirely free. He had at that time a characteristic history of gastric ulcer and a stomach analysis show-

ing a total acidity of 85 and free HCl 65. He refused operation, improved on diet and was not seen again until December, 1912. Then he complained of the same symptoms: about two hours after eating, food began to sour, and belching came on; much gas was brought up; he had severe pain, cramp-like in character; and all this persisted until he vomited spontaneously or induced vomiting or washed out his stomach, which gave relief. At this time the analysis after a test meal gave total acidity 80 and free HC 40. Operation was advised and now accepted. It revealed a small hard ulcer at the pylorus, obstructing its lumen; but also one large stone in the gall-bladder, practically filling up the entire cavity. This man had never had any symptoms pointing to his gall-bladder directly, certainly never any attacks resembling biliary colic. He remains perfectly well since a gastro-enterostomy, with removal of the gall-stone and drainage of gall-bladder. He had a gastric ulcer; but how much of his suffering was due to the gall-stone, and which developed first?

*Fourth*, a woman aged 38 first sought advice in February, 1911, for "stomach trouble," characterized by nearly constant nausea; bloating after eating; much heartburn, belching, gas and general distress after food, but no pain. As the physical examination at that time was negative and the stomach analysis within normal limits, her ailment was classed as a "gastric neurosis." She returned in December, 1916, complaining of a series of attacks of violent pain in her right side and back, under the ribs and radiating under the right shoulder blade, lasting for several hours until she was given morphine for relief. These attacks had never occurred until during December, 1916; but since her previous visit in 1911 her stomach had always caused more or less trouble. Physical examination revealed rigidity and tenderness in the right hypochondrium. Stomach analysis gave a total acidity of 52 and free HCl 24. As attacks persisted with no relief, she was advised to have operation for gall-bladder disease. At this operation the gall-bladder was found thickened and shrunken and containing one small stone. She must have had a chronic cholecystitis for years, which was probably the cause of her gastric symptoms; for no other pathology was found.

Such cases as the foregoing are apparently rare, but may occur

more often than we think. After months or years of quiescence, with no manifestations but reflex ones in the stomach, the gall-bladder may suddenly produce an attack or series of attacks that make the preceding history perfectly clear; but before this time we have no certain proof as to the origin of the dyspepsia. Radiographic plates, however, seem to offer us the most hope; either on the one hand by elimination of such serious organic changes in the stomach as are produced by ulcer or cancer; or on the other hand by demonstrating the presence of gall-stones or of distortions of the pylorus resulting from gall-bladder adhesions. In any event, possibility of gall-bladder disease must always be remembered in every indefinite case of stomach trouble we are inclined to label as "gastric neurosis."

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### A CONSIDERATION OF SYPHILIS OF THE STOMACH —ITS DIAGNOSIS AND TREATMENT

By B. B. VINCENT LYON

Chief of Clinic, Gastroenterological Department, Jefferson Hospital; Pathologist, Methodist Episcopal Hospital; Assistant Pathologist, German Hospital  
Philadelphia

This is a disease of the stomach, caused by the *treponema pallidum* of Schaudinn, which may involve the mucosa, the submucosa or the gastric wall either alone or in combination. It may result from both hereditary and acquired syphilis, but in both types it should be considered a tertiary lesion. While it represents one of the rarer implantations of visceral syphilis, it may yet prove to be far more common than we were once led to believe. About twenty-five years ago Chiari ("Festschrift für Rudolph Virchow," 1891, Vol. II, p. 297) reported 243 postmortems on syphilitic individuals, and found definite syphilitic involvement of the stomach in but two instances (a frequency of 0.8 per cent). More recently Stolper has autopsied 86 patients who have died of syphilis and has found two cases showing definite syphilitic invasion of the stomach. Averaging this total of 329 cases with 4 instances of gastric syphilis, the frequency of this disease from a postmortem standpoint is 1.2 per cent.

It is to be presumed that this estimate will prove far too low, in view of the comparatively large number of cases of gastric syphilis



that have been published in recent years. For instance, William Gerry Morgan (*Am. Jour. Med. Sci.*, March, 1915, Vol. CXLIX., No. 3, p. 392) reports 8 cases occurring in his practice in the past 12 years, the majority of which occurred within the last 3 years, which were diagnosed partly from historic and symptomatic evidence, and partly by X-ray, serological, and operative means. None of these cases came to autopsy. It is manifestly a difficult matter to accurately determine its true frequency. Theoretically we might insist upon the demonstration of definite pathological lesions of syphilis, preferably showing the presence of the specific spirochaeta, but for practical purposes we may rest content in the accuracy of our diagnosis in each suspicious case in which the serological examinations are definitely positive, and in whom syphilitic therapy results not only in a general clinical improvement, but in a cessation of gastric symptoms. If this latter criterion is accepted the frequency of gastric syphilis will be considerably increased. We must remember, however, that syphilis, visceral or otherwise, and other kinds of gastric affections may occur simultaneously in the same patient and each be independent of the other, or cases in whom the gastric condition may be functional, and not organic but secondary to syphilis, as notably seen in the gastric crises associated with syphilis of the spinal cord. Being a tertiary lesion it is far more apt to become manifest during the middle decades of life, and may affect both sexes, and, as stated above, may occur as a result of both congenital and acquired infection.

Pathologically the disease may show any one of the following forms:

1. A diffuse gastritis, involving the glandularis and submucosa.
2. Syphilitic ulcers, single or multiple, frequently assuming ser-piginous forms and having ragged overhanging edges and a smooth base
3. A diffuse infiltration of the gastric wall which histologically must be distinguished from linitis plastica (unless these conditions are one and the same, as many clinicians believe), from a diffuse scirrhus carcinoma, and from a diffuse infiltration of a tuberculous type.
4. Pyloric stenosis.
5. Gumma, which may or may not give rise to a palpable tumor.

From a histological standpoint, the findings are practically those of tertiary syphilis occurring elsewhere. There may be diffuse round-cell infiltration, connective tissue infiltration, frequently a general architectural arrangement in the form of tubercles, often but not invariably furnished with giant cells with the nuclei usually situated eccentrically. There may be areas of coagulation necrosis in the centers of such tubercles, but they show less tendency to the coalescence seen in tuberculosis. There may be endothelioid as well as lymphoid and connective tissue hyperplasia. One of the most constant features is the obliterating endarteritis, which in part may account for the caseation necrosis due to lack of vascularity, and which, too, may furnish a second etiological factor in the production of the ulcer form in gastric syphilis. In some instances the diagnosis can be made beyond dispute by the demonstration of the *treponema pallidum* in tissue differentially stained.

A clinical classification can be readily built up and made to correspond to the pathological one.

*Symptomatology.* The symptoms may vary as widely as the pathological lesion, and in many cases there are no symptoms which of themselves can be considered pathognomonic. Quite commonly we see the symptom-complex of an organic disease of the stomach involving both the motor and secretory mechanism. Aside from the comparatively few instances of motor obstruction due to syphilitic pyloric stenosis, the motor defect is much more commonly due to an extreme degree of atony associated with ectasia. The secretory defect is usually accompanied by the symptoms of a severe atrophic or sclerosing gastritis. Pyrosis is common and is of the type seen in the anacid states; sour eructations, together with the sense of an epigastric lump, weight or pressure, sometimes associated with bloating, the symptoms common to atony, together with the fermentations seen in ectasia.

In the ulcer form one of the early symptoms may be a profuse hematemesis, which is more apt to be recurrent than is common to simple gastric ulcer. In Eusterman's 23 cases (*Am. Jour. Med. Sci.*, Vol. CLIII, No. 1, Jan., '17, p. 21) hematemesis occurred only once. In this form there is frequently pain which is more apt to occur late in the day, bears a less striking time relation to meals than is seen in simple ulcer, and is not so easily amenable to further

food-taking, or to non-specific chemical therapy. There may be constitutional symptoms common to many diseases, such as anorexia, loss of weight, weakness and emaciation. Excessive thirst is not uncommon. In ordinary cases the intestinal functions are properly performed; when deranged constipation is more apt to occur.

*Laboratory Findings.* The gastric analyses much more commonly show a marked subacidity or anacidity with a greatly diminished or absent enzyme activity, which is what one might expect to find associated with the pathological defect of an atrophic gastritis. On the other hand, a few cases have been reported in which the hydrochloric acid content and peptic activity is normal, or even increased. An increase of endogenous mucus is generally the rule. Occult bleeding is frequently encountered both in the gastric filtrate and in the feces. The blood examinations, when diagnostically helpful, usually show a chloroanemia, a moderate leukopenia, with a relative increase in the lymphocytes, and an absolute increase in the eosinophiles. The serological examinations generally yield a definitely positive Wassermann reaction, and is especially reliable when performed by the centrifuge method (Ref. Centrifuge Method to Provide a Uniform Standard for Wassermann Readings. *Am. Jour. Med. Sci.*, Dec., 1914, Vol. CXLVIII, No. 6, p. 885), and when checked by the Hecht-Weinberg Wassermann Reaction as modified by Gradwohl (Ref. Hecht-Weinberg Reaction as a Control over the Wassermann Reaction—a study based on one thousand parallel tests with both methods.—R. B. H. Gradwohl, *Medical Fortnightly*, Oct. 26, 1914).

In cases exhibiting active symptoms the X-ray examination usually demonstrates some definite defect in the gastric outline.

*Physical Examination.* The physical findings may give evidence of a severe constitutional infection, featured by anemia and cachexia, although these may frequently be lacking. Evidence of generalized syphilis may be disclosed in the teeth, gums, tongue or pharynx; in the finding of a generalized adenopathy; in a manifest syphilitic eruption; in visible scars on the genitalia or the scars from syphilitic ulcers on the extremities. Abdominal examination as restricted to the stomach itself may give no diagnostic evidence, but one can frequently demonstrate an atony, dilatation, or both, in the widened area of gastric tympany and the presence of succussion

splashes. In some cases one may imagine the palpatory sensation of a thickened anterior gastric wall. There may be diffuse epigastric tenderness, but even in the definite ulcer cases painful pressure points are often lacking. In some cases there may be palpatory evidence of a gastric tumor, which in emaciated subjects may be visible. This is seen, of course, only in the gummatous forms and in syphilitic hypertrophic stenosis. When a palpatory tumor is evident, it may readily be mistaken for carcinoma, but under observation usually remains quiescent as to size or disappears under specific treatment. Further abdominal examination may disclose evidence of a syphilitic hepatitis or splenitis.

*Diagnosis.* As in some other gastric conditions, the diagnosis may have to be made by a process of exclusion. Particularly is this true in those individuals who give evidence of both a syphilitic infection and a gastric affection, but each independent of the other. In those cases in whom a positive history of a congenital or acquired syphilis can be obtained the diagnosis can be made a clinically sound one if the serological examinations are positive, and there is a cessation of gastric symptoms and a return to normal of the radiographic gastric contour after the exhibition of antiluetic therapy. Further than this the writer cannot do better than quote some of the observations as published in Morgan's paper (*ibid.*):

"1. That the failure to glean from the individual anything suspicious of a syphilitic taint, or an abortion, or failure to have children, or a negative Wassermann does not prove that syphilis does, or does not, exist in that patient.

"2. A diseased condition of the stomach marked by a long duration with changeable symptoms, and which do not correspond to one or other of the well-recognized diseases of that organ, and which resist the accepted methods of treatment, should arouse suspicion of lues.

"3. Tumors involving the pylorus which do not cause stenosis are more often syphilitic than carcinomatous.

"4. Achylia or a low acidity, as occurred in all our cases, is usual in gastric syphilis. And where there is achylia with symptoms of ulcer, one is likely to have an ulcerating gumma or a superficial ulcer on a syphilitic infiltration base in the gastric wall.

"5. Diffuse syphilitic infiltration is usually easily detected by the

palpating fingers because it produces some enlargement of the stomach, which, as happened in some of our cases, may not be readily recognized at operation. This may be true even when the infiltrating mass is to be detected by the roentgen-ray.

"6. A tumor which does not change its size and shape over long periods of observation may be syphilitic, or a tumor which disappears under anti-syphilitic treatment may be presumed to be a gumma."

*Prognosis.* The prognosis is no more grave than is that of visceral syphilis elsewhere, and is usually good if the disease is properly diagnosed and specific treatment is energetically carried out.

*Treatment.* The treatment of gastric syphilis is practically the same as is indicated in any late secondary or tertiary lesion of syphilis, save those which involve the spinal or central nervous system.

The first essential is that the specific therapy should be thoroughly and energetically carried out and the second essential is that it should be kept within the physiological tolerance of the individual patient. Since the introduction of our newer methods of treatment time has not elapsed sufficiently so that we can promise a cure in visceral syphilis. Since the introduction of salvarsan and other forms of intravenous and intramuscular medication in many cases, relapses have been frequently noted. It is yet to be proved whether long sustained treatment with appropriate interruption may finally eventuate in a real cure. Nevertheless, we can be pretty well assured of promising our patients a symptomatic arrest of their disease.

As to the method of therapeutic procedure this often becomes a matter of individual preference, as guided by personal experience. If no syphilitic treatment has ever been given the patient a more intensive and energetic form should be adopted. In the writer's opinion a good deal depends upon the strength of the serological test. This means that every Wassermann that is returned 100 per cent. positive or four plus (for interpretations of difference in nomenclature cf. centrifuge method for Wassermann readings, see author's article, *Am. Jour. Med. Sci.*, Dec., 1914, Vol. CXLIII, No. 6, p. 885) should be quantitatively estimated, inasmuch as it forms such an important check on the effectiveness of our treat-

ment. In one case of gastric syphilis in the writer's experience with a palpable gastric tumor, presumably gumma, the Wassermann reaction was 506 per cent. positive (slightly over twenty plus), and with specific treatment was reduced to 36 per cent. positive (slightly over a plus one), at which time the patient was operated upon for the relief of a complicating duodenal ulcer, to which he finally succumbed.

In some cases spirochetes are locked up in the heart of pathological syphilitic lesions, and on account of the devascularity attendant upon the endarteritis, the syphilitic antibodies may not have access to the circulating blood stream. In such cases the Wassermann reaction may be negative, until a provocative intravenous injection of salvarsan has been given, or may be weakly positive to begin with, with a general increasing positivity under treatment, until a definite point of pathological cure has been reached, when the serological reaction will progressively diminish in intensity.

In all cases of visceral syphilis the three forms of specific chemical therapy, either in periodic courses alone or in combination with one another, will be indicated; namely, potassium iodid, the various forms of mercury, and the various forms of arsenic. Where there is clinical evidence of a palpable gastric tumor, either a gumma or a hypertrophic stenosis, the action of potassium iodid supplemented or associated with the use of mercury, often results in a miraculous disappearance of the objective findings. These two remedies best serve to break down the connective tissue barrier surrounding the gummatous lesions and so liberate the spirochetæ, and give them or their antibodies access to the bloodstream where they can be more effectively attacked by the intravenous injection of neosalvarsan (or preferably?) salvarsan. Potassium iodid should be given in the form of a saturated solution, in a dosage beginning with 10 or 15 drops three times a day, preferably taken in milk, before meals, and increasing the amount given by one drop each dose until the physiological tolerance of the patient has been reached, after which the dosage may be dropped to one-half or three-quarters this amount and continued for interrupted periods of two weeks each for the first year and gradually decreased, if warranted, during the second and third years, with short exhibitions thereafter as long as may be required. Together with this, there should be given mer-

cury, far preferably by deep intramuscular injection in the buttocks, in form of either the soluble or insoluble salts, preferably the former. Such injections should be given daily in courses of six to twelve and then interrupted, to be resumed in a like period, and then to be alternated with intramuscular injections of the cacodylate of soda, beginning with one grain (grams 0.06) and increasing to three grains (grams 0.18). This is the method the writer prefers in such patients as show a low positive serological reaction until the reaction becomes more strongly positive, when the use of intravenous injections of the arsenical group, salvarsan, neosalvarsan, arsenobenzol, is to be begun and given at intervals of a week or ten days until a course of three or four such injections have been made.

The objection to the use of potassium iodid, however useful it may be in the solution of gumma, lies in the fact of its disordering effect upon the gastric digestion. Furthermore, its use should be avoided, or must be cautiously exhibited in tuberculous patients, especially in the quiescent stage of fibroid forms of phthisis, on account of the danger of lighting up this infection.

Likewise the use of the preparations of mercury, either the protoiodid or the biniodid of mercury, or the pill of mixed treatment, when administered by mouth should be deprecated, inasmuch as they not only upset the digestion, but the amount of absorption cannot be accurately controlled. For oral administration the writer prefers the use of calomel, in combination with bismuth subcarbonate or powdered chalk to counteract diarrhea. The advantage of intramuscular injections of mercury are therefore evident. If it is to be administered otherwise, the use of inunctions is the method next of choice. It is needless to state that before beginning such a vigorous use of mercury, the mouth and teeth should be put in a state of oral cleanliness, and thereafter be watchfully so maintained, and should there be evidence of ptyalism or gingivitis, this drug should be discontinued for a short period or its dosage be materially reduced.

In cases in which the Wassermann reaction is relatively high, indicating that the *treponema pallidum* or its specific products have access to the peripheral circulation, the use of intravenous injections of the various forms of arsenic should be begun at once, supple-

mented certainly by the use of mercury, and to a less extent by potassium iodid. Salvarsan appears to be a little more effective than the neosalvarsan, but its comparatively greater difficulty of administration makes it less commonly used. In Trimble and Rothwell's comparative study of salvarsan and neosalvarsan they considered neosalvarsan superior to salvarsan (Ref.: Jour. Am. Med. Assn., Vol. LXVII, No. 27, p. 1984). If there are no contraindications to its use, and if it is well tolerated, an injection should be given every week or ten days until four or five have been made, and then given once a month for the first year, once every second month through the second year, and twice a year thereafter as long as need be. During this time injections of mercury may be given at stated intervals, or a short course of intramuscular injections of the cacodylate of soda, and the periodic exhibition of potassium iodid in small doses (gtts. XXX to LX daily).

The suggestion as to treatment outlined above represents the method that has proven useful in the writer's experience in the treatment of visceral syphilis. It should be thoroughly understood that there can be no syphilitic treatment given by rule of thumb, but a definite plan should be adopted and modified according to the requirements in the individual case. As stated above, the two important essentials are that it should be thoroughly adequate and should be kept within the limits of tolerance of the patient, and finally that it should be maintained until the Wassermann reaction has been consecutively negative for a period of three years, whether this takes five years or the remainder of the patient's life-time to accomplish. Only by this means can we be content with the assurance that the specific condition has been permanently arrested or cured.

Otherwise the treatment of gastric syphilis is purely symptomatic. The dietetic, mechanical, balneological and medicinal treatment is essentially the same as outlined in the management of chronic gastritis.



## A DIAGNOSTIC SIGN OF GASTROENTEROPTOSIS\*

By CHARLES D. AARON

Professor of Gastroenterology in the Detroit College of Medicine and Surgery  
Detroit

Gastroenteroptosis is easily recognized by the use of the Röntgen ray, inflation, auscultatory percussion and transillumination. The author wishes to urge an additional method in diagnosis which suggests a correlative form of treatment.

In cases of gastroenteroptosis, deep, continuous pressure with the ends of the fingers, over the celiac plexus, in the epigastrium will induce pain. (Fig. 1.) The point of sensibility varies in different individuals. To locate it, the co-operation of an assistant is necessary. With the patient standing, the physician applies his fingers in a series of deep pressures until the point of greatest tenderness is found. The fingers are held at this point. The nurse then takes a position behind the patient, passes both arms about him so that the hands, meeting in front, rest on the hypogastrium and lifts the abdomen in its entirety. (Fig. 2.) This relieves the epigastric pain at once, despite the great pressure exerted by the physician at the point of tenderness. When, however, the nurse allows the patient's



FIG. 1.

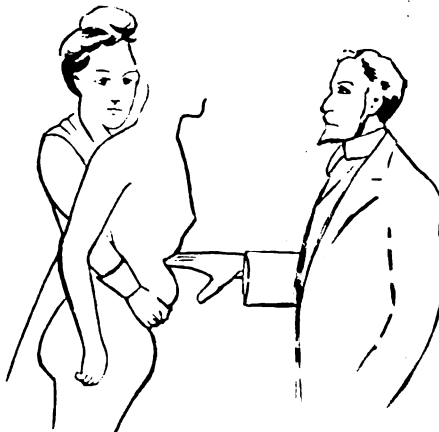


FIG. 2.

\*Read before the American Gastroenterological Association at Atlantic City, April 30, 1917.

abdomen to drop to its former position, the deep pressure continuing, the pain re-appears. (Fig. 1.)

This sign is constant in gastroenteroptosis. In organic disturbances, such as gastric ulcer, carcinoma, etc., the pain under pressure continues even when the abdomen is lifted.

Glénard made clear the necessity of supporting the abdomen by means of a bandage in cases of gastroenteroptosis exhibiting discomfort when the hands supporting the abdomen were suddenly removed. My method confirms this view by the fact that the sensibility to pain through pressure ceases when the abdomen is manually lifted, and equally calls for the employment of a supporting bandage.

Glénard believes that the epigastric pain under pressure indicates a displaced or diseased liver. But sensibility to pain is not always within a definitely circumscribed region; it may be of a diffuse nature. Lennander declares that the intestine, whether normal or diseased, is insensible to pressure, and that pain, when present, is produced through hyperesthesia of the parietal peritoneum. Kelling, again, explains pressure pain by irradiation or reflex diffusion through the abdominal nerve plexus. It is also known that pain may arise from a hyperesthesia of the nerve plexus surrounding the abdominal aorta. The epigastric point of pressure is especially pronounced in nervous derangements of the stomach. My observation, however, leads me to the belief that the pain provoked by the pressure of the fingers upon the epigastrium in cases of gastroenteroptosis is due to the fact that there is a tugging on the nerves in the mesenteries, due to the weight of the abdominal viscera, irritating the celiac plexus. Elsner remarks that the sensibility to pressure pain has its seat in the sympathetic ganglion and nerve plexus of the abdomen; he also believes that the stretching of the nerves in the mesenteries causes pain at the point where the diseased organ connects with the nerve plexus.

The pain on pressure, relieved by lifting the abdomen, reveals an objective sign which I believe to be reliable in the diagnosis and treatment of gastroenteroptosis.

EARLY SYMPTOMS AND DIAGNOSIS OF ACUTE  
INFANTILE PARALYSIS

By LOUIS C. AGER

Brooklyn-New York

The pessimism in regard to the efficacy of the early treatment of infantile paralysis even now so generally present in the minds of medical men is the result of the previous inability to make a diagnosis in the pre-paralytic stage and to the resulting fact that in most instances the full damage to the nervous system is accomplished before any definite treatment is established. The great increase during the past few years in our knowledge of the disease has rendered the pre-paralytic diagnosis quite certain during epidemics, and reasonably accurate in sporadic cases when a proper differential examination is made. It seems probable that in recent years there have been fewer cases of so-called "morning paralysis" than formerly, that is, a prodromal febrile period of some days' duration is usually present. It is undoubtedly true that during this pre-paralytic stage much can be done to mitigate the severity of the symptoms to prevent permanent injury to the central nervous system and even to save life. All this, however, depends upon the recognition of the disease at the earliest possible moment after its onset, and this renders a careful study of the earlier symptoms of the greatest importance.

There is another reason for an early accurate diagnosis, which is less important to the individual patient, but of vital consequence to the community at large. Whether the percentage of "abortive cases" is fifteen or sixty-five, it is the failure to recognize them that permits the rapid spread of an epidemic. There were undoubtedly hundreds of cases in New York in 1916 that received no special examination, and it is unfortunately true that there were many cases in which the tentative diagnosis of paralysis by the family physicians was changed because all the symptoms disappeared in a few days. Many such cases are now being treated for minor paralyses, atrophies and deformities—such as strabismus, subluxation and "flat foot." It is not the sick child in bed—whether isolated or not—but the unrecognized carriers playing with other children, handling their toys, eating the same fruit and using the same dishes, that spread the disease.

For convenience of presentation, the symptoms may be divided into four groups:

1. First, pre-paralytic.
2. Second, the later acute symptoms.
3. Third, sub-acute.
4. Fourth, chronic.

In this paper only the first two groups will be considered at length.

The *pre-paralytic symptoms* are of two kinds; first, those that indicate that a general infection of some kind has occurred, and second, those that result from the beginning involvement of the central nervous system. The first are more or less common to all systemic infections. They are a rise in temperature, general malaise, digestive disturbances, sore throat, moderate glandular swelling, possibly headache. These symptoms vary widely in intensity and taken alone they do not form a sufficient basis for a positive diagnosis even in times of epidemics, as such conditions are present at the onset of any of the contagious diseases, of pneumonia, of typhoid fever and even as a result of improper feeding in hot weather. Nevertheless, in the average case of paralysis these early symptoms are marked and definite. The fever varies from 100 to 105 deg. F. or even higher, and is usually present four or five days, often with a sharp decline to normal at the end of this period. This marked drop is of great importance in connection with the conclusions to be drawn in regard to the results of any form of treatment. In a very large majority of the cases constipation is present to a striking degree, although diarrhea is occasionally observed. Vomiting is the next most frequent symptom, but it is sometimes difficult to determine whether this is due to the disease itself or whether it has been produced by the large doses of laxatives so frequently administered. In many cases, after two or three days of acute symptoms of this kind, there appears an apparent complete recovery. The child is up and about, ready to play and ready to eat moderately. If the temperature is taken in this period, we find that it is slightly above normal, between 99 and 100 deg. F. After 24 to 48 hours there appears an apparent relapse, with sudden rise in temperature, marked irritability and rapid onset of the symptoms which indicate involvement of the nervous system. In

over 75 per cent. of the cases this intermittent period is not present and the symptoms of nervous involvement follow immediately upon the two or three days of acute general infection.

During the past summer so much emphasis was laid upon the upper air passages as the portals of entry of the infection that there was a very general supposition among the medical profession and the laity that coryza was an early symptom of the disease. There is practically no clinical basis for this idea. Occasionally in the latter stages of severe cases, particularly in those of the meningitic type, there is seen a profuse offensive muco-purulent discharge from the mouth, nose and even the eyes just as may be the case in any other overwhelming systemic infection—scarlet fever, typhoid fever, cerebrospinal meningitis. This absence of catarrhal symptoms was noted with surprise by many of the physicians attending the clinics at the Kingston Avenue Hospital, and on more than one occasion it was made the basis for scepticism in regard to the diagnosis in mild cases.

It is theoretically possible that the resisting power of any individual may be sufficient to overcome the infection before any premonitory reactions can occur. Under ordinary circumstances it would not be possible to prove or disprove this in any given case. During large epidemics, however, the simultaneous appearance of numerous infections of this character indicates not only that such cases do occur, but also that they are very frequent. Wickman is generally credited with having proved the existence of this abortive type, but Caverly spoke of such cases in 1894, and earlier writers also called attention to them. The recent epidemic furnished unusual facilities for studying this phase of the disease, and the investigation conducted by Dr. Doty under the grant of the Rockefeller Foundation unearthed dozens of instances in which one, two or three children in a family had had successively acute systemic infection either before or after a typical case of paralysis. It would greatly simplify the comparative study of infantile paralysis if all writers would limit the term "abortive" to these transient cases which give no definite evidence even in the acute stage of involvement of the central nervous system. Unfortunately the term is used very loosely by many writers and is even made to include the cases which Kennedy so graphically described as "Temporary

Paralysis," in which there is definite proof of cord involvement without actual destruction of motor cells; the so-called hyperemia of the spinal cord of Hammond and other writers.

**Pre-Paralytic Nervous Symptoms.**—It is at this stage of the disease that a diagnosis is theoretically always possible, particularly during an epidemic. This implies, however, that the members of the medical profession are open-minded and on the alert to note the earliest evidence of irritation of the nerve centers. It is, therefore, apparent that this second group of pre-paralytic symptoms are worthy of the most careful study. Unfortunately the patients very rarely reach the hospitals during this stage of the disease, but during the past summer, with the physicians and the public on the alert for the earliest manifestations of infection, consultants had an unprecedented opportunity to observe the disease in the earlier stages. Although it is true that even at this stage the manifestations vary according to the type of the disease that may be developing, that is, according to the point in the central nervous system that is receiving the brunt of the attack, the difference lies chiefly in the relative severity of the various symptoms and not in the symptoms themselves. That is, there is always some psychic and motor derangement, some disturbance of the splanchnic nerves and some sensory irritation.

Nevertheless, a brief tabulation of the recognized types of the disease is important even in the earliest stages. The classification of Wickman which is most generally accepted is as follows:

1. Spinal Poliomyelitis; this is the most frequent form of the disease and the one most readily recognized.
2. The Landry or Ascending type; this term is now used rather loosely to describe the cases that are rapidly fatal as a result of respiratory paralysis. It should be confined to the rather rare cases in which there is a definite extension of the paralysis up or down. Not all of the cases reported by Landry were fatal, in one series eight out of ten recovered.
3. The Bulbar or Pontine type in which the cranial nerves are affected, particularly the facial and the ocular.
4. The Encephalitic type producing spastic paralysis.
5. The Ataxic type; this resembles Friedreich's ataxia and the inflammation is probably located in the cerebellum.

6. The Polyneuritic type in which the sensory disturbance is very marked; in some instances this is not accompanied by any paralysis, in others a slight paralysis may follow which is out of all proportion to the sensory symptoms.

7. The Meningeal type in which the symptoms of meningeal inflammation overshadow to a greater or less degree the symptoms of cord involvement.

8. The Abortive type previously referred to.

Psychic Excitation.—Mental irritability is one of the earliest symptoms of any of the acute infections of the central nervous system. It is always seen in the early stages of the various types of meningitis and cerebritis. In the ordinary spinal types of infantile paralysis it usually lasts but a short time followed by more or less Psychic Inhibition. *In ninety per cent. of the cases* this takes the form of somnolence. Children will sometimes sleep twenty out of twenty-four hours. This condition must not be mistaken for coma, as the patient is easily roused to a normal mental condition, will answer questions and take nourishment quite readily, but drop off to sleep almost instantaneously when let alone. This is characteristically different from the dulled mentality of meningitis. It varies from slight and temporary drowsiness to long-continued slumber according to the nature of the case.

In a small number of cases—possibly five per cent.—psychic inhibition does not follow the first stage of excitation. Instead there is a more or less rapid increase of excitability or nervous irritation which may take various forms. Occasionally it resembles the violent delirium observed in typhoid fever, requiring physical restraint of the patient. Wickman is, I believe, the only writer who has described such cases but they have been increasingly frequent during the past ten years or else they have been erroneously diagnosed in the past. They are remarkably like many of the cases of “brain fever” of the last century. A few cases developed neither somnolence nor delirium, but come into a state of extreme hypersensitiveness to their surroundings. They are continually on the alert observing every sound or movement in the room, and refusing to lie down or close their eyes. Even when they appear to be asleep a slight sound will bring them to a cat-like attitude of attention, although they show no special evidence of fear and do not particularly object to examination by nurses or physicians.

**Motor Irritability.**—Hypersensitiveness of the motor tract probably is always present in the initial stage, although it may be of a very transient nature. The most important symptom of this class, convulsions, occurs in about twenty per cent. of the cases. Localized muscular twitching or convulsive movements have been mentioned by various writers. Colliver has described in detail a symptom of this kind which he believes to be of special diagnostic value. Although I have not observed this symptom in any large number of cases it may have been overlooked in many instances, as he suggests. His description is as follows:

“The symptom referred to is a peculiar twitching, tremulous or convulsive movement of certain groups of muscles lasting from a few seconds to less than a minute. The amplitude of vibration is greater than a tremor, not so constant and long as a convulsion, and more regular than mere twitching, yet it has some elements of all of these. It usually affects a part or whole of one or more limbs, the face or jaw, but it may sometimes affect the whole body. The symptom may easily be overlooked in the beginning, as it usually lasts less than a second, and unless the patient is disturbed, does not recur oftener than every hour or so. Later the duration of the spells lengthens to a few seconds, recurring also at shorter intervals.”

Increased knee-jerk and ankle clonus are frequently reported and would probably be found in a very large percentage, if not in all cases, if sought early in the course of the disease. These symptoms soon disappear, however, to be followed by loss of reflexes. Not infrequently, there is an alternate loss and return of knee-jerk for a period of one or two days. According to Wickman, “The loss of the patellar reflex may be the only demonstrable objective sign of the disease.” This reflex may also be increased when the arms are paralyzed, and paralysis and loss of reflex in one leg may be accompanied by an increase in the other leg.

**Spasticity.**—Muscular rigidity of the back and neck is the most important of all the early symptoms of infantile paralysis. In conjunction with the symptoms already mentioned, it is quite diagnostic, and vice versa, its absence at this stage is fairly conclusive evidence that the disease is not present. It must be borne in mind, however, that the muscular spasm of infantile paralysis is of a



peculiar type, as a rule, and that it differs markedly from opisthotonus, as seen in the various kinds of meningitis. It is essentially an antero-posterior spasm, and is best elicited by lifting the patient by the head to a sitting position. It is a subconscious muscular contraction for protective purposes, like the spasm in lumbago or sacroiliac disease. It disappears completely if the patient is placed in an absolutely comfortable position, only to return at the least sign of muscular movement. According to the Rockefeller Institute Report (p. 22), "Of practically constant occurrence are the lesions of the posterior root ganglia." This produces a hypersensitiveness in the inflamed area, and the deep muscles of the back contract to form a protective splint.

Although the spinal muscles are the group most frequently found to be spastic, this symptom is often present to a greater or less degree in other parts of the trunk and limbs. Both the thigh and knee are frequently flexed resembling an exaggerated Kernig's sign and the foot is drawn down producing so-called "foot drop." If the spinal areas controlling the upper extremity are affected, there may result spasticity of the pectoralis group or the deltoid. This stage of spasticity may be so transient as to escape notice entirely, or it may last for weeks and be a source of great anxiety to the physician and patient. The degree and duration of spasticity are usually commensurate with the degree of pain and tenderness—the so-called polyneuritis. In such cases serious damage may be done by mistaking the condition for the true paralytic contraction of the later stage of the disease and attempting to apply forcible extensors by means of plaster or other apparatus. Although this semi-voluntary muscular rigidity is the most important diagnostic symptom of the spinal type of infantile paralysis, in the meningitic type the true reflex spasm is often found, at times producing marked and continuous opisthotonus, and quite a typical picture of cerebrospinal meningitis. The first case that I saw this year (1917) was of this type with the head and heels supporting the body when placed in a supine position. The differentiating symptoms, aside from the spinal fluid findings, were a marked paralysis of deglutition and slight paralysis of one deltoid.

General hyperesthesia.—Pain and tenderness are probably always present in the earlier stages of the disease, but, like the other symp-

toms, they vary greatly in intensity. As previously stated in the brief discussion of the types, they may entirely overshadow the paralytic symptoms or the case may never develop paralysis at all. Apparently these symptoms vary considerably in different epidemics. Wickman, in his report on page 41, implies that it is always a marked symptom of the disease, but in my experience negligible in a very large percentage of the cases. It was very pronounced in practically all the cases that I have seen under the age of eighteen months, but in older children it is not so frequent. In some instances the slightest pressure, particularly on the lower limbs, is exceedingly painful, and even the weight of the bedclothes cannot be borne. True skin hyperesthesia may or may not be present. In extreme cases the gentlest stroking of the leg with the tip of the finger produces great pain. Spontaneous pain is less frequent, but some of the older patients have described backache and pains in the limb of great severity, and the smaller children at times complained of pain without being able to indicate its location.

Bladder and Rectum.—Temporary weakness of the bladder and rectum was described by Heine, and it has been mentioned by practically every writer since his time, yet the symptoms are frequently overlooked. They are present in only a small percentage of the cases, and it is difficult to determine whether they are due to the general psychic inhibition or to actual involvement of the special nerves. These symptoms are usually transient, and the rectal disturbance is not of much importance, but urinary retention should not be overlooked, as is frequently the case. I have very often seen patients with the bladder distended to the umbilicus. Even where catheterization has to be resorted to, the symptom usually clears up in two or three days. In the encephalitic type with continued coma the bladder may require attention throughout the course of the disease.

Sweating.—Profuse sweating has been described by many observers, and in some instances the amount of fluid lost from the body in this way seriously affects the condition of the patient. This symptom is not marked in any large percentage of cases, and is to be distinguished from the normal perspiration of a sick and uncomfortable child during the hot weather, in which the disease usually occurs.

Rashes have been described by many observers with more or less frequency. Dr. Manning (Am. Med., Oct., 1911) lays great stress on this symptom as follows:

"The face is flushed with a mask of white, and the chest, abdomen and back on close inspection show a rubiform or finely petechial rash. In a number of my own cases in Wisconsin, epistaxis had occurred, a fact I have not seen chronicled by other observers. The skin rash, previously mentioned, may be multiform in character, and frequently confirms, in the mother's eyes, her own diagnosis of measles. It is most often overlooked. It may vary from the finest scarlatinal type, a mere blush, to purpuric blotches. The papular and vesicular form observed in six consecutive cases by Dr. Brown and reported in the Dominion Med. Monthly, I have not personally observed." On the other hand, the most careful examination during the recent epidemic gave no evidence of any rashes except the varied eruptions found in any large group of children. Dr. Manning's description strongly suggests a concomitant epidemic of mild scarlet fever or German measles, and the earlier writers on this subject frequently associated paralysis and exanthemata.

The Spinal Fluid.—So much has been written in regard to the spinal fluid in infantile paralysis that little space will be given to it in this paper. Nevertheless, there are one or two points that need to be emphasized. Spinal puncture was performed at least once in every case that entered the Kingston Avenue Hospital. In many cases this procedure was repeated from one to eight times. All cases seemed to show some increase in pressure, although this was not accurately measured. Practically all cases, however, showed the abnormal changes which have been repeatedly described by Draper and others.

The study of 71 cases gave the following figures:

The average cell count 109, 96 per cent. lymphocytes.

The globulin average 2 plus with Noguchi.

In 14 cases the globular reaction was negative.

Fehling reduction was always present with the high average of 83 mg. per 100 c.c. of fluid.

Urea, an average of 24.7 mg. per 100 c. c.

The fluids were none of them actually cloudy, but they showed the ground glass appearance described by Zingher.

Specific gravity showed an average of 1.008.

These figures are averages. The cell count in individual cases may vary from fifteen to several hundred. After the injection of any serum the count usually increases enormously. These changes are quite constant, but there are some exceptions to the rule. In two cases, with all the acute symptoms of the disease including paralysis, the fluid was reported normal in all respects. More frequently the cell count is normal, or shows only a slight increase while the chemical changes are present. In a recent case (1917) of the meningitic type with paralysis, the first report was as follows:

20 c. c. clear fluid.

No increase in cells—a few endothelial cells present.

No organism.

Culture:—negative.

Albumin:— + + + +

Globulin:— + + + +

Fehlings:— + + +

Four days later, after the injection of horse serum (not given with my advice), a second report was practically the same:

15 c.c. clear fluid.

No increase in cells.

No organism.

Culture—negative.

ditto.

And even after four injections of horse serum, there was no increase in cells. This seems to indicate an idiosyncrasy in this patient, as foreign sera usually produce an enormous cell increase. The diagnostic significance of the changes in the spinal fluid will be discussed in connection with the differential diagnosis.

Differential Diagnosis.—In the *Weekly Bulletin* of the Department of Health for November 18, 1916, was printed the following table of the City of New York:

List of Clinical Diagnoses Made in Cases Which Were Found After Admission to Hospitals Not to Be Poliomyelitis.

No illness.....	96
Hysteria .....	2
Uremia and Nephritis.....	1

Tuberculous Meningitis .....	8
Rhachitic Pseudo Paralysis.....	1
Pulmonary Tuberculosis .....	1
Purulent Pleurisy .....	1
Tetany .....	1
Gastroenteritis and Meningismus.....	1
Cerebral Thrombosis .....	1
Epilepsy and Arthritis.....	1
Mentally Defective (Idiocy).....	1
Streptococcus Meningitis .....	1
Purulent Peritonitis .....	1
Intussusception (?) Gastroenteritis.....	1
Dentition .....	1
Congenital Cancaneo-Valgus .....	1
Broncho Pneumonia; Pertussis.....	2
Cervical Adenitis and Cellulitis.....	1
Broncho Pneumonia .....	2
Diph. Paralysis .....	2
Mulnutrition and Spasmophilia.....	1
Seven-Year-Old Case of Infantile Paralysis..	1
Pericarditis .....	1
Kyphosis (Pott's Disease).....	1
Cerebrospinal Meningitis .....	3
Hemiplegia and Syphilitic Endarteritis.....	1
Transverse Myelitis, Specific.....	1
Influenza Meningitis .....	1
Pneumococcus Meningitis .....	1
Cerebral Arteriosclerosis with Traumatic Neuritis of Supraorbital Nerve.....	1
Spastic Paralysis (Congenital Tetanoid Pseudo Paraplegia) .....	1
Chorea .....	1
Bell's Palsy .....	1
Septic Arthritis .....	1
Hemiplegia (Cerebral Hemorrhage).....	1
Measles .....	
Total.....	49

It will be noted that in nearly two-thirds of the cases mentioned no illness was found. It is impossible to estimate what percentage of these children had the abortive type of infantile paralysis and entirely recovered in the few days between report and hospital examination. We do know, however, that in some instances mistakes were made. It is rather surprising that such a small percentage of the total number of cases admitted were found to be some form of meningitis; but this is probably due to the fact that there was very little meningitis in the city at the time. Tuberculous meningitis is particularly difficult to differentiate from polioencephalitis with little or no paralysis. The early symptoms are quite similar. In paralysis the temperature usually runs higher, but not always. The coma is less profound without the slow purposeless movements often seen in tuberculous meningitis, and the patient is quieter and the irregularity of cardiac and respiratory rhythm is not present. Unfortunately the changes in the spinal fluid in these two conditions are very similar, but the more recent methods of examination will usually demonstrate the tubercle bacillus in meningitis.

A few of the other errors are of practical importance, and they will be mentioned briefly, although this paper is not supposed to deal with the paralytic stage of the disease.

Rhachitic pseudo-paralysis apparently reached the hospital in but one instance, which is a great credit to the medical profession. Holt's last edition, page 254, says: "The muscular power in the extremities is sometimes so feeble as to suggest paralysis. We have seen a number of cases in which the symptoms so resembled paralysis, that even expert diagnosticians were unable to differentiate rickets from poliomyelitis except by the electrical reactions, those in rickets being usually normal or exaggerated. In other, cases the symptoms may suggest cerebral palsy of the flaccid type. The muscular symptoms may be marked when the bony changes are slight, and conversely." Unfortunately we now know that the electrical reactions are not as accurate in the differentiation of spinal paralysis as was assumed before the recent epidemic. With intelligent parents the definite history of an acute infection with sudden onset of paralysis ought to clear the diagnosis. It must also be remembered that a very large percentage of the children affected with infantile paralysis are exceptionally healthy.

Similarly the muscular weakness in prolonged and severe attacks of persussis and chorea resemble infantile paralysis on casual examination, but a mistake in diagnoses seems unnecessary. Apparently all the cases of Mongolian idiocy in the city were at one time or another during the epidemic mistaken for paralysis. Two cases were admitted to the Kingston Avenue Hospital, and the diagnosis was accepted on the spinal fluid findings in conjunction with a history of acute illness. Possibly the changes in the fluid are present in all cases of Mongolian idiocy, and it would be well to have this point determined.

In the examination of infants two conditions that have repeatedly been mistaken for infantile paralysis in the past must be excluded. These are scorbutus, and epiphyseal separation due to congenital syphilis. The fact that the polyneuritic type of paralysis is frequent in infants complicates the diagnosis. Some years ago I saw a case which had been called infantile paralysis by an orthopedic surgeon clear up in two days under the use of orange juice. When scorbutus has reached the stage of pseudo-paralysis the other symptoms of the disease are always present. In either of these conditions a gentle and painstaking examination will show the actual seat of the pain, and will prove that there is no real paralysis.

#### CONCLUSIONS

1. It is quite possible to make a diagnosis in most cases of infantile paralysis before paralysis has occurred, provided a careful examination is made.

2. The essential diagnostic symptoms are:

- An acute febrile attack.

- Sensory and motor excitability.

- Semi-voluntary antero-posterior spinal rigidity which relaxes to a great extent if the back muscles are properly supported.

- The customary changes in the spinal fluid.

# THE DIAGNOSTIC PROGNOSTIC AND THERAPEUTIC VALUE OF LUMBAR PUNCTURE IN SPINAL INJURIES

By HAROLD NEUHOF

New York

Unquestionably, the importance of lumbar puncture in the diagnosis of spinal injuries must be appreciated by many, yet evidences of the recognition of its true value cannot be found in the literature. Diagnostic lumbar puncture has proven of such signal aid in the First Surgical Service of Bellevue Hospital that we now employ it as a routine measure in all cases of suspected or of evident spinal injury. Its value lies, of course, equally in the demonstration of the presence or of the absence of blood in the cerebrospinal fluid. This test has so aided in clarifying our views concerning the indications for operation for spinal injuries that the presence of large quantities of blood (and evidence, upon repeated punctures, of continuation of the bleeding) comprises one of the exceedingly few indications for operation for recent spinal traumata.

In other cases, the disclosure of blood in the spinal canal was the only evidence that vaguely suspected spinal injuries were in fact vertebral traumata with cord compression. Two such instances were seen. An impressive one was that of a man who entered the hospital in a profoundly shocked, semi-comatose condition, with the history of a fall from a considerable height. When seen by me immediately after admission to the hospital, a contusion over the lower dorsal vertebræ was noted, but signs of injury to the cord could not be elicited. Lumbar puncture disclosed the existence of active subdural bleeding. When the patient recovered from shock, the evidences of an incomplete injury of the lumbar cord were evident.

The groups of cases in which the presence of blood may be of unusually great value as indication of the existence of spinal injury (with cord compression in most instances) are:

1. Suspected spinal injury in individuals suffering from acute or subacute alcoholism.
2. Suspected injury in patients in coma or in confused mental states.



### 3. Injuries suspected in cases of hysteria.

Blood in the spinal canal is not of such definite diagnostic value in cases of skull injury, in which associated spinal injury is suspected, for, as is well known, blood in the cerebrospinal fluid may be the result of a cranial lesion. However, the presence of large quantities of fresh blood in the spinal canal when there are some signs of spinal injury, points to the latter as its source.

Small amounts of blood admixed with cerebrospinal fluid naturally indicate less active hemorrhage. The precaution should always be taken to collect the spinal fluid in two or three test tubes, in order to eliminate trauma made by the puncture needle as the source of the blood.

The lumbar puncture demonstration of the absence of an intradural hemorrhage is, as has been said, of equally great value, not only in the diagnosis and in the indications for treatment, but also for the immediate and ultimate prognosis. The existence of clear cerebrospinal fluid eliminates one of the very few indications for operation for recent spinal injury. This is well shown in the following case:

The patient was admitted to Bellevue Hospital a few hours after having been flung against her spine in a street car accident. There was some evidence of a spinal cord injury, with steady progression, so that, at the end of 36 hours, there was complete loss of power and sensation in the legs, and absent reflexes. The case seemed one in which there was progressive hemorrhage about the cord and for which operation was indicated. Lumbar puncture revealed absolutely clear fluid and operation was withheld. The reflexes of the lower extremities reappeared soon after, and the subsequent course of the case was that of a mild injury to the cord with super-added hysteria.

The therapeutic value of the withdrawal of subdural collection of bloody fluid is evident, particularly in those cases for which operative procedures are not indicated; yet lumbar puncture for such purposes finds no advocates. If the spinal cord manifestations of recent injuries are due to compression or irritation by fresh blood, it is clear that removal of a part or all is desirable, not only for the relief of the immediate symptoms, but also to minimize the after effects of the hemorrhage about the cord. Conceding that

only a part of the blood can be removed by lumbar puncture, even that is, I believe, better than permitting all to remain. As a further step, it may prove feasible to attempt to remove by washing some of the remainder. Up to the present time this has been practised in only one case. The immediate results were striking, but the final outcome is unknown, for the patient was removed to his home a few days after.

The patient was admitted to Bellevue Hospital with a fracture of the lower dorsal spine, profound sensory loss over the lower extremities up to the level of the 12th dorsal segment, marked paresis of both legs, increased reflexes, Babinski phenomenon and other indications of pyramidal tract involvement, and retention of urine. The catheterized specimen of urine showed the evidence of an advanced nephritis so that operation was absolutely excluded except for the most stringent indications. Lumbar puncture revealed considerable quantities of fresh blood in the spinal canal. As much as would flow freely was withdrawn. Then repeated washings with sterile water were made until the return flow was no longer very deeply colored. Within two days the power in the legs had very greatly improved, the sensory loss was of a much slighter degree, and the patient voided voluntarily. It is, of course, impossible to say how much improvement might have occurred without this procedure, but the immediate result is certainly suggestive.

There is much more to be said of the value of lumbar puncture in spinal injuries, chiefly in reference to the prognostic significance of clear cerebrospinal fluid. Enough has been adduced, however, to demonstrate that it is as important for the diagnosis of spinal injuries as it has proven to be for the diagnosis of meningitis; I believe its diagnostic value in spinal injuries to be far greater than in spinal tumor or other chronic intraspinal lesions. Furthermore, it may prove to have definite therapeutic value, especially if combined with irrigations in spinal injuries accompanied by active bleeding. The procedure should, therefore, be employed in every recent spinal injury sufficiently to lead to the suspicion of injury to the spinal cord or its membranes.

DIAGNOSIS AND PROGNOSIS IN FRACTURES OF THE  
BASE OF THE SKULL

By F. E. BUNTS

Cleveland, Ohio

It has long been the custom to speak of fractures of the base of the skull as injuries of the greatest moment, and yet it is a well recognized fact that only in exceptional instances is it the fracture that is the serious lesion.

It is, of course, the associated injuries, the lacerated brain, the hemorrhagic extravasations, edemas, the contusion, compression or meningitis that determine the immediate prognosis, and it is a realization and understanding of these conditions which must guide our treatment. In other words, a simple fracture of the base obviously requires no treatment, save rest, and quiet, and so it is to the complications that we must give our attention. Before doing so, I would like to consider the more striking symptoms of basal fracture, which are, it seems to me:

First. Bleeding from the nose, ear, pharynx, into the scalp over the mastoid or in the neck below the occiput, and hemorrhage into the ocular conjunctiva. Any one or all of these may occur, and no fracture of the base of the skull be present, but if we can rule out local contusions or lacerations, there can be but little doubt as to the injury, and often a very good estimate of the location of the injury may be made since, local lesions being eliminated, epistaxis and subconjunctival hemorrhage commonly signify fracture in the anterior fossa, hemorrhage from the ears, middle fossa, hemorrhage from pharynx and ecchymosis about ear or in tissues of the posterior neck, the cerebellar fossa.

Second. Cerebrospinal fluid. One of the most positive signs of fracture of the base, especially if its discharge from the nose or the ears follows prolonged hemorrhage.

Third. Nerve injuries. These are very common in fractures at the base, and where peripheral injuries and, in some cases, cortical lesions of the brain are ruled out, become strong presumptive evidence of fracture. Thus we have injuries to the seventh or facial with coincident facial paralysis, injuries to the olfactory with disturbance of smell, and diplopia and paralysis of muscles of eyeball following involvement of motor oculi nerve.

Fourth. Sinus injury. A fracture across the middle fossa may injure both the carotid artery and the cavernous sinus, so that an arterio-venous aneurism results, and symptomatic of this condition we have a pulsating exophthalmos; injury of the cavernous sinus with consequent thrombosis may also cause a marked protrusion of the eyeball, so that the ball of the eye may be practically forced out from between the eyelids.

Fifth. Cerebral symptoms. These are not dependent upon the fracture alone, for the fragments are not widely separated and can scarcely be depressed so as to cause compression, but they are due, when present, to the associated lesion, so that we often have the familiar symptoms of compression from hemorrhage or edema, concussion, contusion and laceration.

Sixth. Pulse and respiration. Slowing of the pulse and Cheyne-Stokes respiration, which are so often associated with severe fracture at the base in some stage of its progress, are not, of course, symptomatic alone of this lesion, and since they are present in such a wide variety of diseases and injuries, must be given but little weight in making a diagnosis.

Seventh. Spinal fluid. This, when obtained by lumbar puncture, often appears bloody, but as laceration of the brain or rupture of artery or vein may give this same finding, it is only to be regarded as slightly corroborative of fracture.

It would appear from a review of these symptoms that eliminating external and local injuries, which might simulate them to a limited extent, the most positive evidences which we have of fracture of the base of the skull are, the hemorrhages already alluded to and the discharge of cerebrospinal fluid from the ears, nose or pharynx, and it seems to me that the next most important sign is evidence of nerve involvement, especially of the seventh.

I cannot dismiss the subject of diagnosis without referring to the use of the X-ray examination. It is impossible to say just what proportion of fractures of the base of the skull will be revealed by the X-ray, but we must be prepared for failure in a very considerable number of instances, and the reason is not alone due to the normal irregularities of bones at the base, or to the fact that the fracture is often a mere fissure, but also to the inherent difficulties in taking the negative. It must often be taken from several

different angles before the fracture is revealed, and the patient is often so restless, or delirious or the breathing is so heavy that it is almost impossible to get a satisfactory X-ray examination. Fractures in the posterior fossa show with considerable constancy; next in frequency of good results is the anterior fossa, while fractures through the middle of the base probably will not show in more than 50 per cent. of cases. When the fracture extends into the vault, as it often does, it is much more readily and constantly detected.

Now, what is the prognosis to be expected when, aside from the hemorrhage or discharge from the ear, or when there is a nerve injury as the result of a fracture of the base, no other symptoms are present? I believe it is ordinarily good. The nerve impairment may be more or less permanent, but frequently it is entirely recovered from and the greatest danger that we have to fear is infection and the development of meningitis. Obviously primary operation could not be expected to do any good in this, the simplest form of basal fracture, and indeed a large proportion after the first symptoms of concussion have passed get well without any serious disturbance whatever, and nothing but the later development of compressive symptoms, whether from infection or otherwise, would justify operative interference.

Laceration of the brain is a lesion commonly associated with basal fracture. It may be so great as to cause sufficient hemorrhage to produce compression symptoms, but in the absence of these, we must rely chiefly for its diagnosis upon evidence of nerve injuries and upon the obtaining of a bloody fluid from a lumbar puncture, and, too, I think, upon evidence of cerebral irritation, particularly delirium or insanity. From my own experience clinically, I would say that none of these symptoms demand operative interference, there being nothing certain or definite to operate for, and there is nothing, so far as I am aware, as yet, to make us feel that the ultimate results so far as cerebral disturbances would be in any way benefited by an operation of any kind.

Associated fracture of the vault with brain injury or compression will, I believe, always demand operation in the hope that removal of spicula or bone, elevation of depressed portions of skull, evacuation of blood clots or macerated brain, and establishment of

drainage and providing for passive or inflammatory edema may save the patient and prevent some of the more serious forms of brain sequelae. And yet, in looking over my case records, it is just this class of cases in which operation has seemed absolutely imperative, that fatalities have been so numerous as to be almost without exception, and the reason is not, as I apprehend, because we operate or because we don't operate, but because the nature of the injury has necessarily been so serious as to render a more favorable outcome practically hopeless.

Compression of the brain is a more or less common accompaniment of fracture at the base, and may be due to edema from venous obstruction, as in traumatic sinus thrombosis, to hemorrhage, or to pressure of the displaced fragments of the skull itself. Compression may be an early complication, or it may come on several hours or even days after the original injury, and though we have, as yet, comparatively few statistics upon which to base a definite line of treatment, it seems reasonable to consider very seriously the question of decompression operations in all these cases. The degree or the severity of the compression symptoms often depends quite as much upon the location of the hemorrhage, for instance, as it does upon its amount, so that the removal of the clot is not the most important indication, rather it is by timely interference to so relieve the pressure as to prevent an early and fatal outcome. Though the clot may sometimes, as has been done, be successfully removed, it is more frequently inaccessible or so spread out on the base of the brain as to be impossible of removal. The inaccessibility of a basal clot makes its exact location relatively unimportant from an operative standpoint. Of course, in the presence of an associated fracture of the vault, with pressure symptoms and possible hemorrhage, the site of operation is readily decided upon, but under other circumstances probably the transmuscular operation in the temporal region will be the most favorable site, not only for the extensive removal of bone, but possible location of a clot.

Where the symptoms develop gradually ophthalmoscopic examination may give the first serious danger symptom in the beginning development of choked disc, and in its presence I do not believe that operation should be delayed. To this end early ophthalmoscopic examination should be made, for, if operation is to be of

value, it must be performed before the late stages, when complete choked disc and medullary compression with Cheyne-Stokes respiration and slowed pulse have occurred. That not all severe cases of compression demand decompression operation may be illustrated by one of my recent series of brain injuries, in which the gradual supervention of epileptiform convulsions and finally coma, with slowed pulse and stertorous breathing, determined me upon doing a decompression operation, but preliminary to this I did a lumbar puncture, removing a considerable amount of spinal fluid, and his symptoms began to improve so soon afterward that no further operation was done, and a perfect recovery, mentally and otherwise, resulted.

I have the records of 33 of my own cases of fracture of the base of the skull. In most of these the diagnosis could be readily decided upon from the presence of the cardinal symptoms alluded to. In some it was cleared up or corroborated at autopsy.

The column devoted to operation or nonoperation does not mean a decompression operation, for while that was undertaken in one instance, the operation was usually for an associated injury, such as fracture of the vault. In view of the almost uniformly bad showing made for the results in operated cases I simply wish to call attention to the chief symptoms present, which emphasize a previous statement made, that the operation was undertaken in exceptionally bad cases with associated injury of skull and brain were present. I do not wish to shirk any possible criticism of technic leading to such a fatal array of cases, but to state that it is my belief that the operative disclosures in all of them made it evident that they were primarily fatal. The criticism which I have to offer is that surgical judgment should have shown me beforehand that a favorable outcome was impossible, and yet it often seems that it is in the face of the seemingly hopeless things that we refuse to acknowledge that there is no chance, and operate because the patient will surely die if we don't.

Of the 33 cases here tabulated 16 died and 17 recovered. Of the 16 fatal cases 6 were operated upon. In only two was a decompression operation performed, and one of these was done while operating for an infected mastoid. The remaining operations were either exploratory skin incisions or elevation of depressed fractures of the vault which had occurred at the same time.

In the cases that recovered a reference to the table will show that the symptoms were severe in many cases—evident compression of the brain having been recovered from without any operation whatever other than a lumbar puncture in one case.

While some difference of opinion still exists among surgeons regarding the question of operating in all cases of fracture of the base, I believe that those of greatest clinical experience are inclined to agree with Frazie and Eisenbrey that when once the circulation and respiration begin to fail, the vasomotor or respiratory center was unable to recover its tone or function whether or no decompression operation was practiced. And this corresponded to their experimental findings that decompression of the brain had absolutely no deterring influence upon the ill effects of intracranial tension in serious cases. It is probable that only too often the fatal pressure is not due to hemorrhage or clot, but to a general edema of the brain which, owing to the rigid separation of the falx and tentorium, prevent a decompression from producing anything more than a local relief, and this is often followed by such a marked bulging of the brain through the opening and pressure upon the edges of the bone defect, that it seems as though the brain would sustain decided injury from local pressure at this point.

I have no doubt but that had all these cases of recovery without operation been operated upon by a decompression operation and have recovered, one might have been influenced to regard many of them at least as due to the operation.

In conclusion I would express it as the result of my clinical experience:

First. Simple cases of fracture of the base of the skull without severe laceration of the brain practically all get well without operative interference.

Second. The greatest cause of danger in these cases is from septic meningitis and operation cannot prevent this complication, but rather adds to its probability.

Third. The bad cases, complicated by a bursting fracture of the skull with an extensive laceration of the brain, practically all die.

Fourth. It is in this class of cases that operations such as removing spicula of bones, crushed brain, etc., are most frequently resorted to, but usually without avail.



Fifth. Distinct compression symptoms, coming on immediately after the injury without obvious evidence of extensive brain laceration would probably be deserving of decompression operation, for they might be due to hemorrhage and be susceptible of relief or arrest.

Sixth. Late symptoms of compression with beginning choked disc might be due either to hemorrhage or edema, and should be subjected to immediate operation, though in the one case in which I had an opportunity to follow this plan, operation and subsequent autopsy showed no clot, very slight hemorrhage, but extensive laceration of the brain, not only in the lowest frontal convolution near the site of fracture, but in the occipital region far from the fracture the laceration of the brain was even greater than in the frontal.

Seventh. There are insufficient available statistics as yet to show that decompression operations hasten the recovery after fracture of the base, or lessen subsequent liability to cerebral disturbances.

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### PAIN IN THE BACK

By WALTER S. REYNOLDS

New York

Pain in the back is a symptom referable to many different diseases. To determine the cause of the pain is often difficult and not infrequently requires considerable time and patience before the matter is made clear. One of the conditions which is becoming more frequently recognized as a cause of pain in the back is urinary calculus. This is undoubtedly due to the aid derived from cystoscopic and X-ray examinations. There are few cases where the information obtained by means of these diagnostic aids is not reliable. Having an accurate means of determining whether such a foreign body is present or not, the question arises, is it possible to decide whether such an examination is necessary, what are the indications or guide. An examination of the urine, while not always reliable, will in the vast majority of instances be an accurate guide, and in the absence of abnormal substances in the urine, especially pus or blood, we may assume the absence of calculus. It must not be forgotten, however, that in exceptional instances a normal urine may be present where calculus exists.

The presence of calculus is usually announced by pain, not infrequently the only symptom. There is nothing in the character or location of the pain to indicate where the disturbance originates, but in this paper, for the sake of brevity and to avoid confusion, I shall confine my attention to ureteral calculus.

When we come to consider the symptom of pain, its cause and the frequency with which it occurs ureteral calculus, we find considerable difference of opinion regarding these questions. In some cases, it has been stated that pain may be altogether absent. It is also said that calculi have remained unsuspected until revealed by autopsy. I should be inclined to doubt the correctness of these statements regarding pain. It hardly seems possible that there might not at some time have been evidence of the presence of a foreign body of this sort, either by pain or other symptoms which, of course, might have been so slight as to have caused little inconvenience or disturbance, and consequently might be easily overlooked. In my experience cases are not infrequent where pain is not severe, and the other symptoms correspondingly mild. These are the cases which are most apt to be overlooked for some time, and the pain in the back treated for lumbago or similar condition until perhaps a sharp attack of colic gives rise to a suspicion as to the cause of the trouble. We must not allow only typical colicky pains to be a reminder of calculus. All pains in the back must be considered as possibly due to calculus.

It has been stated that the passage of a calculus does not of itself cause pain, but that the pain is caused by intrarenal tension due to plugging of the ureter. (Bevan, *Trans. Am. Urolog. Ass.* Vol III, p. 397). While this may be true in cases where the stone is of such shape or size as to fully block the ureter and prevent the escape of urine, it has been the experience of all urologists, I believe, to see many cases where the urine may be observed by cystoscopic examination to pass from the ureter in a manner apparently normal.<sup>1</sup> Another writer (Bryan, *Surg. Gyn. and Obst.*, Feb., 1911) says: "The pain of stone itself is that produced by pressure on the ure-

<sup>1</sup>The effect of ligation of the ureter was discussed at a meeting of the Southern Surgical and Gynecological Ass'n 1911, and opinions seem to have differed regarding pain following such an accident. This would seem to be analogous to the complete obstruction of the ureter by a calculus.

teral wall, the pain of tension. It is also dependent upon the composition of the stone, the oxalate of lime apparently produce the worst attacks, and the uric acid the least severe."

The apparently diverse views held by the writers already quoted may be supplemented by that of a third, who assumes that both factors, either singly or together, may be responsible for the pain. The writer, Osgood (Trans. Am. Assn. G. U., Surg., Vol II, p. 283), says: "The severity and character of the pain have doubtless, in great part, a direct relation to the traumatism done by the movement of the calculus or the distension of the pelvis and ureter above the partial or complete obstruction, and the back-pressure produced thereby upon the kidney, which secretes under difficulty, and, we may reasonably presume, becomes hyperemic or congested, with consequent distension of its capsule. A cramp contraction (tonic spasm) of the muscular coat of the pelvis and ureter, combined with traumatism of the mucous membrane, accounts for the ureteral colic. The character of the calculus has much to do with the severity of the pain and the traumatism produced. A sharp or rough calculus, as many calcium oxalate and phosphatic calculi, the size of a pin-head, may produce the most agonizing colic, while a smooth-surfaced stone, nearly as large as a cherry, may be borne in the pelvis or ureter, without severe pain throughout life."

As a matter of fact, these explanations are probably based on supposition. Bevan cites one case to substantiate his theory. He says that he has also seen other cases similar in character, all of which has led him to the conclusions which he expresses. His case seems convincing so far as that particular case is concerned, but does not explain the cases where the flow of urine is apparently not obstructed by the calculus.

At present I believe we are not in possession of sufficient evidence to warrant any positive statements, but rather to assume, as Osgood does, that the pain may be due to the character of the calculus, to the obstruction caused, by the inflammation produced by the foreign body or a combination of these.

The question may be asked whether or not it is of any practical interest to determine the cause of the pain. I believe it is for the reason that if we know the cause we may be in a position to better control it than by the administration of morphin, which has been

our chief reliance heretofore, but which, as is well recognized, does not, as in pain elsewhere, have the same anodyne effect. In an article by Macht (*Jour. of Pharm. and Exp. Therap.* Vol. IX, No. 3), in speaking of the action of morphin in ureteral colic, he says: "It has been shown that morphin, as regards its local action on the ureter, increases its spasm, and will therefore aggravate the condition." In a case which I reported (*Med. Rec.*, Dec. 14, 1912) it was shown that the injection of oil relieved the colic and hastened the passage of the stone. I shall briefly review that case as reported adding the sequel to it. I select this case because it illustrates the great difference in the character of the pain and because of the interest attached to the subsequent history of the case.

Case: A man 32 years old suffered several attacks of pain, accompanied by frequency of urination and bloody urine, quite typical of colic due to calculus. X-ray confirmed the diagnosis. Cystoscopic examination showed urine being passed in somewhat lessened amount from the affected side. By the use of oil injections and dilation the calculus was finally passed into the bladder to be expelled during urination. The injections of oil served to relieve very markedly the pain which before treatment frequently compelled him to stop work. This calculus was irregular in shape, with a small sharp spine at one point. So much of the case has been reported. The man remained well for three years. During that time he had been careful as to his diet, and had drunk water plentifully as I had instructed. When I saw him again he had been having for a short time some backache which he said resembled his previous trouble, but not nearly so severe, and there had been no attacks of colic necessitating his stopping work. The urine was pale-yellowish in color, with only a faint turbidity and no signs of blood. On cystoscopic examination the bladder was found to be normal, the ureters apparently discharging the urine in a normal rhythmical manner. The right ureter catheter passed freely and easily, but the left was obstructed at 7 c.m.—a point nearly the same distance from the ureter opening as the calculi found to be lodged on the opposite side three years previously. After some little manipulation the catheter was made to pass the obstruction. On three subsequent occasions the same procedure was carried out except that a bougie was substituted for the catheter. A few days after the last treatment a calculus was passed, but unfortunately it was lost.

This case illustrates the two types of calculi cases. The first attack giving rise to unmistakable colicky pains with urinary symptoms equally characteristic. The second attack might easily have been overlooked had the man not had a previous experience. Neither the pain nor the urinary symptoms would have given rise to suspicion as to the cause of the trouble in a hasty examination, unless he was on the lookout for such cases. The case is interesting on account of the recurrence of calculus, but on the opposite side.

It would be difficult to say in the first attack how much of the pain was due to obstruction and how much to the character of the stone. There probably was a certain amount of damming back, as the urine was not escaping as freely as it normally should. The sharp spine was, I have no doubt, a factor of considerable importance. In the second attack it is a question how far the character of the stone influenced the pain. As the stone was lost, there is no means of determining this point.

I have referred to the injection of oil and the effect it had in relieving the pain. This could, of course, be due only to the lubricating action of the oil. If the pain is due to the obstruction and damming back of the urine, other means must be taken to afford relief. In Bevan's case, already referred to, this was accomplished by drainage of the kidney by means of a lumbar incision. Lewis (*Am. Urolog. Assn.*, 1909) reported a case where by means of an indwelling ureteral catheter drainage was satisfactorily accomplished. He also used the catheter for the purpose of injecting alypin solution to relieve the pain. This was not a case of calculus, but of pyelitis. It serves to show how it would be possible to secure drainage providing the catheter could be made to pass the calculus.

No attempt has been made to discuss the treatment of ureteral calculus in this paper. I have been interested only in the question of pain as a symptom of calculus.

In conclusion, I would say that in cases of pain in the back we should not overlook the possibility of the presence of calculus and that pain, in my experience, is always present.

Urinary analysis, X-ray and cystoscopic examination are to be relied upon as a means of diagnosis.

## FACTORS IN THE PROGNOSIS OF SYPHILIS OF THE NERVOUS SYSTEM

By EDWARD LIVINGSTON HUNT

New York

In the present day when so much time has been given to the diagnosis and the treatment of syphilis of the nervous system, too little attention has been paid to the important element of prognosis. After all, what the syphilitic wishes to know is whether he is going to get well, and after all, what we medical men are here to do is to get the patient well, or as near well as possible. It is, therefore, of interest and of the greatest possible practical importance to consider the subject of prognosis in syphilis of the nervous system.

There are many factors which have to be taken into account in judging this condition. In the first place, it is absolutely necessary to find out the character of syphilis of the nervous system. I mean by that that it is necessary to locate the exact pathological lesion. There are five pathological conditions which occur in syphilis of the nervous system: (1) the involvement of the meninges, (2) the involvement of the arteries, (3) the involvement of the brain substance (4) the involvement of the cord substance and (5) syphilis of the peripheral nerves. To reach an intelligent prognosis it is necessary, therefore, to distinguish exactly what particular part of the nervous system is affected, and to what extent.

To diagnose the exact involvement of the nervous system, it is necessary to examine a certain secretion of the body. This means that the patient must undergo a lumbar puncture and have a small amount of cerebrospinal fluid examined in the laboratory. The value of this examination is twofold—it not only assists the clinical examination in reaching a definite diagnosis, but it also corroborates the clinical examination; in some instances it goes further than the clinical examination and itself establishes a definite diagnosis, a definite type of involvement of syphilis of the nervous system. The examination of the cerebrospinal fluid, however, plays a further part in the conclusion of this subject. It forecasts the future, establishing a definite prognosis. The test which is of especial value in this particular point is the one known as the colloidal gold test.

This, as is now well known, consists in the precipitation of certain substances in the spinal fluid and the change in color of the contents of a series of tubes when small amounts of spinal fluid are added to a gold solution. According as these tubes change color, and according to the shades of the colors, it is possible to designate with considerable certainty and accuracy the exact type of syphilis of the nervous system, the exact portion of the nervous system which is affected, and further than that, the more than probable, the almost certain, part of the nervous system which will become involved as the disease progresses.

In reaching a prognosis of syphilis of the nervous system, it is now becoming more and more important to ascertain, if possible, just what strain of spirochete is infecting the human organism. It has been found that certain types of spirochetes will bring about an acute and serious infection, while others will tend to a more subacute and less violent infection. It has also been shown that certain types will give rise to one form of syphilitic involvement, while others will give rise to a wholly different type. Thus, one syphilitic afflicted with general paresis may bring about in all those who become infected from him, only paresis. Again, a still different factor is the particular reaction which each individual displays toward the attack of the syphilitic virus. Thus, one individual may be able to withstand the attack and wholly resist it, while a second may be able to resist only in a mild way, and become affected to a limited degree, while a third may be unable to resist at all, and may at once develop an acute, severe, and fulminating type of syphilis.

A factor which ought to be considered in the prognosis of syphilis of the nervous system is the length of time in which the patient has suffered from the infection, that is to say, the period which has elapsed from the initial lesion to the date of diagnosis. It is well recognized that certain types of syphilis can remain latent for a long time and yet yield to treatment, while still other types may, in a very short time, make tremendous inroads on the individual. I mean by that that it will depend on the resistance displayed by the individual and on the type of the spirochete pallida—that is to say on the strength of the individual and the strain of the bacterium.

An important point, and one which is not sufficiently thought of, is the strength and persistency displayed by the Wassermann test

on both blood and spinal fluid after a certain period of time has elapsed, and after a certain amount of treatment has been tried. Thus, it will be seen that a Wassermann, which at the end of six intraspinous injections changes only from a four plus to a three plus, will show less chance of improving than a Wassermann, which at the end of two or three treatments has changed from a four plus to a two plus. The prognostic value of the Wassermann test is far greater than is that of the cell count. The value of the cell count is almost wholly in diagnosis, and to my way of thinking, the value of a Wassermann on the blood is greater in prognosis than in diagnosis. A practical point in the consideration of this subject, and one to which not sufficient attention has been given, is the part played by alcohol. It must never be lost sight of that a patient who has been taking a large amount of alcohol will show for some days afterward a negative Wassermann.

A point which may seem trivial, and yet one which may assume great importance, is the character of the laboratory. By the character of the laboratory I do not mean to cast any aspersions on laboratories, but rather to call attention to the fact that in all laboratories involving examinations of syphilitic fluids the personal element must enter, and unusual care must be exercised. Furthermore, as there is no standard for any laboratory to go by, and as there is no recognized standard for the different tests which are made on the spinal fluid, it follows that discrepancies and differences may occur. It has by no means been infrequent in my experience to find two laboratories disagreeing on a blood Wassermann, and I am very sure that if more of us will have two separate examinations made, we will be surprised to find how frequent are the errors. This will be further emphasized when you realize that a proper Wassermann examination of the blood means a quantitative examination and not a qualitative examination. No Wassermann examination of the present day is correct unless it is reported in the several dilutions. It must be reported in 1 c.c., in .5 c.c., and even .2 c.c. The reason for this is that in certain dilutions the Wassermann is negative, while in others it still remains positive. Therefore, a prognosis of value cannot be made until the Wassermann examination has been reported in the smallest possible dilution of the spinal fluid.



The clinical examination is another factor. It is well known that the more profound types of syphilis, as a result of treatment, cause few clinical changes. In these cases the pupils and knee jerks are rarely altered, whereas in the more superficial and acute types of syphilis, treatment brings about considerable change clinically, as is evidenced by the great improvement which occurs in tremors and speech. Under the heading of clinical examination I want to include not only the physical signs, but also the mental. In paresis and in some cases of cerebrospinal syphilis there is an alteration in judgment, orientation, memory, and all the higher faculties.

In giving a prognostic opinion of syphilis of the nervous system, one must consider the character of the salvarsan and the methods of its administration. It will make a difference whether we use salvarsan, neo-salvarsan, or the Canadian preparation of di-arsenobenzol. It will make a difference as to the method of administration, whether it is given intravenously or intraspinally. It is of considerable importance to know that we are administering a product which is standardized, and which is neither diluted nor fraudulent.

Again, one must consider the method of administration of other drugs, such as mercury, iodid, and arsenic, but especially mercury. There is no doubt that the administration of mercury by mouth is absolutely useless, and a waste of time. It is also true that the administration of mercury hypodermically, when it results in the formation of hard indurated lumps, is equally useless. The formation of such indurations simply means that the mercury has become locked up in the tissues, and will be of no practical value to the patient.

The environment of the patient during the treatment is important. Those patients who are obliged to continue their daily occupations, and subject themselves to anxiety and worry will not do as well as those who go to a sanitarium or live in the country.

Another factor is the information given by both ophthalmoscope and sphygmomanometer. Thus, an ophthalmoscopic examination which shows a beginning pallor of the optic discs would forecast a different result from one which showed normal discs. A marked increase in blood pressure is important in prognosis. Those cases which have an unusually high blood pressure cannot stand as radical

treatment as those which present a more normal figure; therefore cases that present an abnormal blood pressure should be a factor of some importance in making a prognosis.

Consider the condition of the kidney, as illustrated by the urine examination. There is no doubt that a kidney which already shows signs of breaking down will be less favorable for future treatment than one which is normal. Such a kidney will be unable to stand excessive doses of either mercury or arsenic, and will require slower progress, milder dosage, and therefore give less brilliant results.

### DIAGNOSIS AND PROGNOSIS OF CEREBRO-CEREBELLAR DIPLEGIA

By L. PIERCE CLARK

Chief Consultant in Neurology and Psychiatry to the New York City  
Children's Hospitals and Schools, Randall's Island  
New York

Congenital cerebro-cerebellar diplegia is the term used for a combination of symptoms dependent upon cerebellar agenesis or to some form of injury to the cerebellum, at the time of or before birth, the exact nature of which has not yet been determined. The disorder presents varying types of symptoms which may be grouped roughly into those of flaccid and flaccid-spastic palsy, shading into the general mildly spastic state. The latter associated with cerebellar symptoms has been carefully described by Batten<sup>1</sup> as congenital cerebellar ataxia. The severest type of cerebro-cerebellar diplegia is embraced in my group of cases described four years ago.<sup>2</sup> In the pure cerebellar ataxia type there are hypotonia, dysmetria, gross incoordination, astasia, abasia, dysarthria, occasionally dysphagia and often complete inability to sit up because of extreme trunk ataxy. In the extreme involvement of the fore-brain the foregoing cerebellar symptoms are present together with mutism and the lowest grade of idiocy. In this latter group type there may or may not be evidence of involvement of the pyramidal tracts. In the mixed types of the association syndrome (cerebro-cerebellar type), there may be either a slight degree of spasticity in certain parts combined with hypotonia, or flaccidity in other parts of the body.

The first clinical recognition of the syndrome without recognizing

the essential cerebellar character of it is to be found in descriptions of cerebral palsies, such as that of Freud,<sup>3</sup> in which there were cases described as showing flaccidity, etc. As stated before, the first effort to take the cerebellar symptoms, that of the asynergies, out of the ordinary picture of cerebral palsies, was performed by Batten in his essays.

The ordinary flaccid type is fairly illustrated by the following clinical picture:

CASE I is a girl of 6 years, a twin, born at the mother's first pregnancy. The birth was thought to be three weeks premature. She weighed  $4\frac{1}{2}$  pounds. The twin sister weighed  $3\frac{1}{2}$  pounds. Two subsequent children (boys) in the fraternity weighed  $8\frac{1}{2}$  and  $10\frac{1}{2}$  pounds respectively. Our little patient had an umbilical hernia, which disappeared without operation. She was an extremely passionate child, and her training has been about equally divided between conduct and social training and motor training. She was not able to sit up at the time the other twin was walking. Prior to this observation nothing unusual was noticed. The flaccid musculature of the back caused the parents to visit an orthopedic surgeon; she was then given an elaborate system of training by removable braces. She commonly lay on her back; the lower extremities bent considerably at the knees and hips strongly abducted and rotated outwardly. The legs could be easily over-extended and remained so without position. It was soon noted that the musculature of the whole body was flaccid and incapable of ordinary motivation. There was no atrophy or electrical changes. Further examination showed there was no muscular resistance and the different segments of the limbs could be maintained easily in very arbitrary positions without pain. This extreme hypotonia had naturally an intimate relationship to the absence of voluntary motion and station. At the end of two years the back muscles were sufficiently "hardened" so she was able to sit up, and was pronounced cured. At this time, when sitting and standing were encouraged, a cerebellar ataxy was noticed and incoordination of all extremities was marked when any movement was undertaken. At four years of age, some eighteen months ago, she was able to walk, but did so with the most remarkable ataxy and incoordination, comparable to a mechanical doll. All movements were dysmetric and arhythmic.

Articles which she picked up would fly out of her hand as though catapulted from her fingers. She walked best with a wide base; even then she could hardly enter an ordinary doorway without striking herself on either or both sides of the door jam. In spite of the fact that she had the repellant leer of a low-grade mental defective and could not talk, by ordinary performance tests she showed no diminution in native ability so far as it was possible to judge. At the present time, eighteen months since the first examination, it is obvious that the ataxy, incoordination, dysmetria and hypotonia are steadily diminishing. The child is able to speak a fair number of sentences, although the speech appears to be without conscious direction. She is only learning slowly "how" she speaks. She is surprised to hear the sound of her own voice, and only after a great deal of practice is she able to produce this larger group of formerly spontaneously produced sentences. With a little assistance she feeds, dresses and undresses herself, and engages in all the ordinary everyday activities. Her general intelligence is that of her years. Her interests are far beyond her general appearance. There is even more discrepancy in her general appearance, and her performance ability than that often noted in *cerebral diplegics*—one is often surprised at the degree of general intelligence such children really possess.

In brief, we have here a case of cerebro-cerebellar diplegia in which the more prominent defect is cerebellar in character. Aside from the delay in ability to speak and a slight retardation in the development of the mental processes, the cerebral defect bids fair to be overcome within the next few years of training. It is in such cases one may expect the training-out of cerebellar defects in the motor asynergies to give the most brilliant results in mental development.

The milder grade of the spastic type of this disorder is shown in the following:

CASE II is that of a little girl now eleven years old. She was brought to me first when seven years of age, suffering from epileptiform convulsions and a "peculiar lack of coordination." A third and a second maternal cousin were epileptic. A maternal aunt was neurotic and subject to depressions. The mother and her brother were not of a nervous type, but were very slow in games and sports which required agility.

Our patient was the first child in a family of two. Her birth was beyond term (2 weeks); there was a face presentation, and delivery was instrumental. She was a blue baby, and the cord was about the neck. She weighed  $7\frac{1}{2}$  pounds. She was not a particularly passionate child, but exhibited a few tantrums. She was super-sensitive in regard to taking food and becoming accustomed to clothing. Dentition was slow and difficult. She was constipated at times, but had no convulsions until five years of age. At about two years it was noticed she was backward in walking. She had only been able to stand with difficulty at 12 months, and this was maintained with much wavering. There was some rigidity in the right leg and arm, and a slight degree of flaccidity on the left side, especially in the leg.

Our patient had a double internal strabismus, which was more marked on the left. The epileptic attacks were not classic; there was no tongue-biting or voiding. They were usually in the left arm, focal in onset, although at severer periods they became general attended by loss of consciousness, and a short coma followed. There have been no attacks for the past four years.

At the time she came to me for examination at the age of seven, psychometric tests showed a mental retardation of a little less than two years. This retardation has been lessened somewhat in the past four years. Examination showed a fairly well developed child for her age, in good physical health. There was an irregular implantation of the teeth. The palate was low, with broad torus. Swallowing was difficult. The speech was indistinct, inclined to be monotonous and of the scanning type. She was markedly incoordinate in all movements. A marked ataxy was shown in standing and walking; marked dysmetria was present also in the upper extremities. Attempts to run increased the incoordination. The reflexes were normal. A general neurological examination was otherwise found to be negative.

In the four years intervening between the first examination and the present time, our patient steadily gained in physical and mental development. The dysmetria has greatly disappeared; the incoordination and ataxy are only markedly noticeable during excitement and fatigue; when overtired she is quite awkward, and the right leg shows considerable spasticity. A slight degree of spas-

ticity is present in the right leg when she is at rest. Attempts at rapid coordination of different segmental parts of an extremity are performed poorly, and are very irregular (adiadokokinesis).

This case is one of cerebro-cerebellar diplegia in which the cerebral involvement has much improved inasmuch as careful teaching has permitted a wider range of manual training to be applied to the cerebral education per se. It is a good clinical illustration of Batten's type.

Undoubtedly many more cases of congenital cerebro-cerebellar diplegia of both mild and severe types pass unrecognized in the general designation of cerebral palsy, where gross incoordination, defects of speech, and locomotor defects loom rather large in Little's syndrome as well as in other descriptions of cerebral disorders entailing either feeble-mindedness or diplegia, or both. The automatic and natural outcome of mild grades of cerebro-cerebellar defect is fairly illustrated in the following:

CASE III is a man 32 years old, single, and is preparing to reenter a medical school in which several years ago he contracted a mental torticollis, and because of which he had discontinued medical study. He was born after an instrumental dry delivery. He was a blue baby for several hours. Physical development was delayed, and he did not talk and walk until the middle of his second year. He then walked with an uncertain straddling gait. Various devices were tried to overcome the weakness in the legs. There was a marked spasticity in the left leg, but a mild degree of hypotonus in the right. The spasticity and flaccidity, awkwardness and incoordination were slowly overcome, and at the age of sixteen it was fairly trained out by an elaborate system of physical gymnastics. His speech, which was at first delayed, was scanning, syllabic and over-precise, and this still endures in greater part. The speech defect, the physical awkwardness, slowness and arrhythmic jerkiness of segmental movements of the extremities are more than vestiges of the former cerebro-cerebellar diplegia. As a child of three or four, he could not perform any of the complicated or difficult coordinated acts, and had a "tremor" in the hands—all of which became marked whenever he became excited. His rate of mental acquirement was similar to that which he showed physically, and if his mental development depended upon rapid physical co-

ordination, it was further handicapped. Recognizing these gross physical and mental faults, the boy was encouraged by his father to take up all sorts of physical sports, which he did. In time he became an expert long distance bicycle rider.

One would at first think this man was of the simple minded class, but on closer observation one finds he is only naive and peculiar, somewhat lacking in discrimination and judgment, and possesses a general defect in the essential understanding of life. He is not agile mentally, nor easily adaptable to different social situations. There was a retardation in mental development all through his adolescence, but the very persistence with which he now studies and strives to win his belated goal—a medical education—shows the essential primary endowment of native ability was not defective.

The prognosis of all these varying types depends, of course, upon the combination of symptoms presented. It may be fairly stated that when the forebrain is damaged to such an extent that the mental state is no longer to be classed as retardation, but shows mental arrest or marked imbecility, such children usually never recover either from their ataxia or from their defect in mental development. Inasmuch as the prognosis in an individual case of the less severe types depends upon our being quite sure that the mental state is one of simple retardation or mental arrest, much of the definiteness of prognosis cannot be determined until one has made a very decided effort to train out the cerebellar symptoms or force the forebrain to take up vicariously the functions of the cerebellum. This being the case, the kind and character of treatment is of great importance.

While the principles of treatment really embrace mental as well as motor training, the primary point of approach in any treatment must be directed toward overcoming the motor defects. This may be embraced under general headings; first of all, the removal of the segmental incoordination. In such treatment the child is taught to appreciate by actual manipulation the flexion, extension, abduction and adduction of the different segments of the extremities. The training is not so very dissimilar to that of Frenkel's<sup>4</sup> system for tabetic ataxia, and his principles for training trunk coordinations are of the utmost moment in these cases. However, when one has obtained results in removing the segmental defects, there still remains the general motor training of using these simple, isolated con-

tractions of segments into purposive acts in which all the segments of an extremity are coordinated harmoniously. When this has been fairly well inaugurated, one may undertake more definite principles of motor training, such as are laid down in the Montessori, Seguin and kindergarten systems, with music, rhythmic games, and sports, and under the guise of play the child is further taught to develop coordination.

In the majority of cases there is an ataxia of speech which needs close consideration. This may range all the way from a state of over-preciseness of syllabication to a marked dysarthria as seen in disseminated sclerosis or in the gibberish of the aphasic. The process of training here must be similar to that used in teaching the deaf and dumb. With this motor incoordination fairly taken care of or removed, there still remains to be handled many of the temperamental defects which almost invariably go with these cases. This fault makes these children very difficult to handle. The disciplinary system of nursery ethics has much to do toward helping the child to develop out of his motor and mental disorder. This type of training is similar to that which I have laid down for epileptic children.<sup>5</sup>

In conclusion it may be said that the essential principle of training for these children is to use (1) a general training in games, sports, and a broad concrete system of physical and mental education; and (2) at the same time employ a special training in physical gymnastics to teach the use of segmental movements of all sorts, and finally (3) to give the child a thorough understanding of the rhythmic continuity of all the different segmental movements combined in a full purposive act, be it simple or complex, or fully coordinated with other bodily acts (diakonesic).

The training should be carried out for a number of years under the supervision and direction of a capable trained nurse-teacher, upon whom the success of the work must largely depend. I trust these general remarks upon the training system which I have employed on a series of cases for the past four years may not only be of service in this type of cerebellar disorder of children, but that it may serve some purpose in encouraging a more persistent and careful training of many children suffering from cerebral palsies and the simpler types of feeble-mindedness, where cerebellar symp-



toms of incoordination which are comparable to those most exquisitely shown in these cerebellar diplegics may also be found to exist. Perhaps the principles here laid down may be found of service even in adult cerebellar disorders in which similar symptoms are in evidence if the lesions in such diseases are not too destructive or too rapidly progressive in type.

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## THE DEGENERATE: BORN DELINQUENCY AND CRIMINOLOGIC HEREDITY

By J. VICTOR HABERMAN

Instructor in Psychotherapy and Clinical Psychology, College of Physicians  
and Surgeons, Columbia University; Visiting Physician,  
Department of Neurology, Vanderbilt Clinic

New York

There is a type of individual much in mind these days through popular interest in the wake of eugenics, prison reform and other momentary fad-movements, who, nevertheless, presents a grave sociological problem in which not only society and criminology, but clinical psychology, must take deep and serious concern. This is an individual whom the psycho-clinician has frequently to pass on, with whom the courts are familiar, for his is a most frequent presence among delinquents and criminals, and whom the layman himself has sized up and labeled *degenerate*.

What is a *degenerate*, and what are degenerate family lines, and

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**Special Articles**

A STUDY OF VASOMOTOR EFFICIENCY

By GEORGE W. NORRIS and FRANKLIN F. LANE.

Philadelphia.

That the functional efficiency of the circulation bears a definite relation, in direct proportion, to the health of the individual has been demonstrated by many experimentors, but just which diseases cause the greatest loss in efficiency and which recover the soonest, has not been dwelt upon to any great extent.

With this in view, at the suggestion of Dr. George W. Norris (who is now "somewhere in Europe," and whom the author was to assist in writing this paper by gathering the data), blood pressure readings were made on a series of cases in the Pennsylvania Hospital.

There have been several methods devised by different authors to measure the vasomotor efficiency; some complicated and others fairly simple in their technic. Most of these methods are based on sound theoretic principles, but they are not absolutely accurate, due to the numerous sources of error. They are, however, sufficiently accurate to form a basis upon which to work. One great source of error is the personal equation. This may be cut down to a minimum, however, if, in a long series of cases, one person makes all the readings.

The value of any test for the functional efficiency of the circulation is threefold. First, from the standpoint of diagnosis; secondly, from the standpoint of prognosis; and, thirdly, from the standpoint of the efficacy of the therapeutics.

The cases for experiment were divided into three classes as follows:

Class A: Those with cardio-vascular diseases.

Class B: Those with some constitutional disorder.

Class C: Those in comparative health. As all of this class were hospital cases, few individuals in really good health could be obtained. The results from this class cannot be of as much value as the first two, except for comparison.

This group, however, is the most interesting from the three standpoints of diagnosis, prognosis and therapeutics. In the first two classes there are many methods of diagnosis, without a test for vasomotor efficiency. The prognosis and the value of the treatment of a case may be told by the disappearance of edema, dyspnea, cyanosis and other pathological findings and the reappearance of normal conditions.

In class C, however, come the cases which though apparently well, yet lack the stamina and punch to carry through a day's work, and those who are always complaining, yet in whom no pathological condition can be found. There is something wrong somewhere, but the finger cannot be placed on it.

These cases are usually of two types, both having low blood pressure; latent syphilis and undemonstrable tuberculosis.

While a definite diagnosis cannot be made by blood pressure and pulse readings, yet by the low blood pressure and low percentage of efficiency of the circulation, the underlying causes of the continued weakness can be recognized, the proper treatment instituted and the prognosis noted.

An overtrained athlete can easily be detected by the thin pinched face, the dry shiny skin, and the haggard look about the eyes. But one who is just on the verge of overtraining and just beginning to go backward is difficult to recognize.

A very good idea of the physical fitness of the athlete, or non-athlete, can be easily determined by a test for the vasomotor efficiency. This test is simple and easily carried out by anyone. It does not require any special knowledge or training. A blood pressure apparatus, a watch, and a scale on which to grade the findings are the only necessary implements, and it takes less than five minutes to perform the test and calculate the result.

The Crampton test was selected as the most desirable on which to form a basis for these experiments.

This test, as do the others, depends on the manner in which the pulse rate and blood pressure respond—quantitatively, qualitatively and temporarily—to definite changes in posture. In rising from the recumbent to the erect posture blood pressure tends to fall as the result of gravity. In health this tendency is automatically regulated. In a healthy, vigorous man this change in posture gives a rise of blood pressure amounting to 8 or 10 mm. of mercury and an increase of pulse rate from 0 to 4 per minute. This increase may result from increased vasomotor tone, from increased cardiac effort, or from both. With these facts in mind, Crampton devised the following table to test vasomotor efficiency by observation of the pulse rate and blood pressure responses to a change of posture.

#### VASOMOTOR TONE CHANGES

1. In health, the vasomotor tone is increased and the blood pressure raised 10 mm. Hg. on changing from the recumbent to the upright position.

In disease (mental or physical) blood pressure either remains the same or falls more than 10 mm. Hg. It may be considered normal to fall 10 mm. Hg.

2. Heart rate. In health, no change on change of position.

In disease, increase to as much as 44 beats per minute.

This change in pulse rate may take the place of change in blood pressure.

Decrease of 1 mm. Hg. = increase 2 pulse beats.

#### PERCENTAGE SCALE.

(Vasomotor Tone)

Blood Pressure

% = % of Efficiency of Vasomotor System.

HEART RATE INCREASE	INCREASE BLOOD PRESSURE					NO CHANGE BLOOD PRESSURE	DECREASE BLOOD PRESSURE				
	+10	+ 8	+ 6	+ 4	+ 2		- 2	- 4	- 6	- 8	-10
1-4	100%	95%	90%	85%	80%	75%	70%	65%	60%	55%	50%
5-8	95	90	85	80	75	70	65	60	55	50	45
9-12	90	85	80	75	70	65	60	55	50	45	40
13-16	85	80	75	70	65	60	55	50	45	40	35
17-20	80	75	70	65	60	55	50	45	40	35	30
21-24	75	70	65	60	55	50	45	40	35	30	25
25-28	70	65	60	55	50	45	40	35	30	25	20
29-32	65	60	55	50	45	40	35	30	25	20	15
33-36	60	55	50	45	40	35	30	25	20	15	10
37-40	55	50	45	40	35	30	25	20	15	10	5
41-44	50	45	40	35	30	25	20	15	10	5	0

NOTE: In case of increase in pressure higher than + 10 add 5% to the + 10 column for each rise of 2 mm. Hg. in excess of 10.

The readings are made with the patient in the recumbent posture, by counting the pulse until two successive quarter minutes are of the same rate. The rate for one quarter is then multiplied by four and recorded. The systolic blood pressure is then taken by the auscultatory method. The patient then stands and when the pulse has steadied and two successive quarter minutes are alike the rate per minute is recorded, the systolic pressure is again taken and recorded. By comparing these results with the Crampton scale a numerical statement of the function may be obtained.

#### *Class A*

Ten cases were studied all with signs of decompensation. When compensation had been established for three or four weeks they were again studied. After eight weeks there were four remaining in the hospital due to complications such as anemias, aneurism or nephritis, and these cases were again recorded.

Before compensation the average vasomotor efficiency according to the Crampton scale was .....62.5%

After compensation had been established for three or four weeks the average was .....93.6%

After four weeks more of treatment the four remaining averaged .....97.0%

The lowest of the ten before compensation was .....35.0%

The highest was .....75.0%

After compensation was established the lowest was .....80.0%

And the highest was .....100.0%

#### *Class B*

Fifty-eight cases in all were studied. This group consisted of the following convalescent patients:

Diseases	Number of Cases
Pneumonia .....	10
Acute Rheumatic Fever .....	5
Acute Bronchitis .....	5
Gastric Ulcer .....	5
Acute Plumbism .....	4
Typhoid Fever .....	3
Chronic Nephritis .....	3
Diseases of Thyroid .....	3

Acute Appendicitis, post-operative .....	3
Acute Tonsillitis .....	2
Empyema .....	2
Tuberculosis of Kidney and Bladder .....	2
Sapremia .....	1
Lung Abscess .....	1
Perirectal Abscess .....	1
Necrosis of Jaw .....	1
Cervical Adenitis .....	1
Sarcoma of Wrist .....	1
Diabetes Mellitus .....	1
Mucous Colitis .....	1
Myasthenia Gravis .....	1

All of the above cases were not studied until they were ambulatory and had from one to three months' treatment.

This series averaged.....	45.8%
The lowest was .....	10.0%
The highest was .....	67.0%
After one to four weeks' additional treatment those remaining (about 40) averaged.....	81.0%
The lowest being .....	40.0%
The highest being .....	100.0%

### *Class C*

As stated before, none of these patients could be classed accurately as healthy, vigorous subjects except in a comparative way. They were all apparently strong.

In all, seventeen cases were studied, consisting of the following:

Diagnosis	Number of Cases
Fractures .....	6
Hernia .....	3
No pathological condition .....	3
Conjunctivitis .....	2
Rheumatoid Arthritis .....	2
Old Infected Hand .....	1
These averaged .....	79.3%
The lowest was .....	55.0%
The highest was .....	100.0%



*Summary*

Class A (Cardio-Vascular) :	
Before compensation .....	62.5%
After               " .....	93.6%
After eight weeks of compensation .....	97.0%
Class B (Convalescent infections	
4-12 weeks in bed) :	
As soon as out of bed .....	45.8%
After 1-4 weeks' more treatment.....	81.0%
Class C (In apparent general physical health) :	
Only one reading for each patient .....	79.3%

## CONCLUSIONS

1. Cardio-vascular conditions do not seem to affect vasomotor efficiency as much as do the prolonged infections and other constitutional disorders.

2. That the latter group of diseases affect the system more profoundly is shown by the length of time to recover their vasomotor tone.

3. After compensation has been established vasomotor efficiency is surprisingly good, while after an acute infection it is surprisingly low, although the individual seems to be in excellent physical condition.

4. Although class C is composed of comparatively healthy individuals yet the results indicate that their vasomotor efficiency is higher or lower in proportion to their general health. Taken as a whole, the findings of this group were higher than either of the other two, these being a nearer approach to the normal. This would seem to indicate the value of the Crampton test in:

(a) Diagnosis of physical fitness.

(b) Furthermore, it seems to have some prognostic value, inasmuch as readings taken at different periods seem to indicate whether the individual is returning to normal or not.

(c) Finally, the method furnishes a clue as to the efficiency of the therapeutic measures employed.

## RACHICENTESIS AND SPINAL FLUID

BY JOSEPH H. BARACH

Pittsburgh, Pennsylvania

## I

Quinke, formerly of Kiel, now of Frankfort, is the man whose name deserves first mention in a general consideration of the topic—lumbar puncture.

While it is 2,100 years since Hippocrates tapped the ventricles for the relief of hydrocephalus, it is hardly twenty-five years since any serious inroads have been made upon the diagnostic and therapeutic possibilities of lumbar puncture.

We are very far, even now, from a complete understanding of the possibilities and limitations of this avenue of medical investigation, but it is good to know that we are certainly on the way to a clearer conception of the problems herein involved. Perhaps the Future will prove that our present diagnostic criteria were not valid, that our therapeutic attempts were futile and were not without harm, nevertheless the Future will have to admit that the Present was its stepping-stone, and that it served willingly and well.

It is since the writings of Quinke that lumbar puncture has become a useful adjunct to diagnosis, and as the literature upon the subject shows, an amazingly large amount of investigation has been carried out in the last few years.

The spinal fluid has been carefully studied in many diseased conditions, and from various viewpoints. The clinician, the pathologist, the bacteriologist, the serologist and the bio-chemist, all have taken an interest in the changes to which this fluid is liable, and each has contributed his share to the sum total of our present knowledge.

It would be a difficult task to offer a complete presentation of this subject. Much of the work of the past, in this, as in other fields of investigation, has been of a negative character. A considerable proportion must be set aside because of faulty methods employed in the investigations, and still another portion must be set aside because the findings of some investigators have not been verified by the experience of others. But these limitations are inherent in the advance of all phases of medicine; and yet out of it all, a crystal-

lization finally takes place, which forms the fixed facts upon which we depend in our daily work.

Such a presentation is the aim in the following pages; to present the experiences and ideas which have passed the purely experimental stage, and may be said to have earned for themselves the recognition of having a definite clinical value.

*Anatomical Consideration.* Without devoting too much space, it will serve us well, if we refresh our memory upon a few important anatomical features which enter into our consideration. The central nervous system, contained within the cranium and vertebral canal, is enveloped by the membranes of the brain and cord. These are from within outward, the pia, the arachnoid membrane and the dura mater. Between the pia and the arachnoid membranes is the cerebrospinal fluid.

This pool of fluid constantly bathes the brain and spinal cord, and aside from its other functions, acts as a buffer substance between the delicate nerve tissues and the osseous structures which contain them.

*The Pia* is a delicate membrane made up of fine areolar tissues supporting a plexus of blood vessels. It covers the brain, dips down into the convolutions and fissures, and just as intimately surrounds the spinal cord.

*The Arachnoid* overlays the pia; it is a thin, loose-fitting membrane, which envelops the entire brain and cord. It passes over but does not dip down into the sulci between the convolutions and fissures.

Connecting the pia and arachnoid is a fine web of connective tissue strands. These fibers are short where the pia and arachnoid are in close contact, and they are long where the pia, having dipped into a sulcus of the brain, is at some distance from the arachnoid.

*The Subarachnoid Space* is the space between the pia and the arachnoid. It comprises those indented areas on the convex and under surface of the brain and cord which contain the cerebrospinal fluid. The subarachnoid space communicates with the general ventricular cavity of the brain by an opening in the inferior boundary of the fourth ventricle, the Foramen of Majendie.

*The Dura Mater* is the outermost membrane which covers the brain and cord. It is a dense inelastic fibrous membrane, serving as

to what degree is there sense and substance in the talk about "born degenerates," "born criminals," "moral insanity" and innate antisocial tendencies—for in this heritable connection the facts are eminently important for us—and segregation, castration, etc., in the hope of ending such dangerously tainted stock? In brief, what is the problem of degeneracy, and what are the facts in criminologic inheritance?

Let us first of all sift the nomenclature and diagnostically differentiate our terms, for in the literature the name "degeneracy" has been taken much in vain, and has been so variously used and misused that no two writers appear to imply precisely the same thing.

Morel<sup>1</sup> was probably the first to give special survey to this term in his "law" of inherited decay, in which, as he thought he had discovered, the stock declension through convergent inbreedings of taint gradually intensified—*degenerated*—going from bad to worse, so that where in the earliest generation, one might have nervous temperaments and ethical inferiority, the next showed severe neuroses and alcoholism, the third psychoses and suicides, the fourth or fifth idiocy and aphasias, the line here dying out through sterile end-members. This "law" of Morel's, however, was not substantiated. Whether, indeed, such families occur at all, is most questionable.

In spite of the failure of the theory, the name *dégénéré* was still retained by French writers and applied, without sharp definition, however, to quite another heritable condition, much as the German "Entartung."<sup>2</sup>

By way of elucidation let us fancy (even though our metaphor will not prove most consistent) the heritable mental elements or personality-constituents in the germ-plasm as building stones which after fecundation multiply and form the human character. If the stones are evenly blocked and cuboidal, the final building proves plumb and firm. This the normal individual. But should some damaged, uneven or badly formed stones have come into the mass—from some poor mating (usually it results from convergent psychopathy)—the edifice which results is uneven, unfirm, in short, defective, with rifts and breaches in the stories—an edifice which might hold out, but, on the other hand, under strain, might collapse in part or fall to pieces. This is the *dégénéré* of the French (the

*Entartet* of the Germans). Such individuals harbor a heritable potential tilt to psychopathy, or have quite outspoken evidence of faulty mind. Where abnormal *variability* and *lability* are pronounced, the literature speaks of *instabiles*,\* where an unevenness of character is the chief flaw, then of *déséquilibrés*. Still others, through inability to profit by expedience, are the *indisciplinés* or *incorrigibles*. If such individuals are normal in intelligence, they are *dégénérés supérieures*, if feeble-minded, *dégénérés inférieures*. The *degenerescence* in them is synonymous with taint, Belastung or 'noxus hereditas or paternus'.

But degenerescence or hereditary taint occurs in quite dissimilar and other pathologies too, and the term is therefore a poor one for this group. What distinguishes its members is an abnormality in the mental dynamics, in the *psyche*, an abnormality in mental reaction, either an over-reactionability or inadequacy of reaction, or again, unusual tendencies in ideation. These abnormal thought processes—this *psychopathy*—manifests itself, in a measure, after the pattern of the various neuroses and psychoses, but only very rarely goes over into the latter. It constitutionally affects the individual, however, colors his mental make-up and *characterizes* him. Hence Kraepelin termed these individuals "abnormal characters" or "abnormal personalities." J. L. A. Koch, in the field of remedial pedagogics, applied the name "psychopathic inferiority," from which we get our anemic "constitutional inferiority." The most virile and appropriate caption is Ziehen's "Psychopathic Constitution"†—a name now very extensively accepted on the continent.

In still another connection do we come upon the term *degeneracy* in the literature. Ziehen, namely, in his arrangement of the psychopathic types, designated one as "the generally degenerative psychopathic constitution," or "the hereditary-degenerative psychopathic constitution."<sup>8</sup> These cases occur on the basis of especially

\*Kraepelin calls these the "Haltlosen."

†See author's "The Psychopathic Constitution," *Med. Rev. of Rev.*, March, 1912, and literature there cited. The chief mental characteristics noted in these individuals are lability of mood, weak will, uncontrollable affectivity, dysphoria, lack of concentration and of attention, abnormal phantasy, abnormal thought impellences, etc. Ziehen has divided them into the following types: the affective, obsessive, depressive, hysterical, neurasthenic, paranoid and the degenerative.

strong hereditary\* taint, mostly, indeed, convergent besides.† In this type not only the psychic, but also the somatic abnormalities are numerous (stigmata degenerations‡). A full description need not be given here—nor of the variations within the group—as this again is not the individual we mean to bring into light, the ‘degenerate’ of the lay mind and the courts.

Who, and what, then, are our lingo *degenerates*? They are a set that belong rightfully enough, for the most part, under the last named rubric, but are only an especially differentiated type of the larger variegated group of hereditary-degenerative psychopathic constitution, for many of this larger group have little or nothing to do with the conditions the public mind stigmatizes as “degenerate.” A composite description would sound as follows:

Conspicuous in the early childhood of these individuals are tendencies that go sharply over the bounds of normality. So, for instance, there is a history of precocious sexual awakening, with either mere sexual hyperesthesia or actual abnormal practices. Such children may masturbate excessively, or transgress with other children—sometimes of the opposite sex, sometimes of the same. In later years these abnormals may be guilty of seductions or the misuse of children, or homosexuality, incest and even sexual acts with animals. In some, the sexual plays a large and manifest part; in the larger number, however, it is just episodic or a background concomitant.

\*Occasionally the same symptomatology and picture is given by organic brain disease (or trauma) in very early childhood. Ziehen also mentions the fact that he has come upon severe cases showing an exquisite picture of this type (where even several brothers and sisters were subjects of degenerative psychoses) in which no hereditary taint nor any other etiological factor whatsoever could be discovered. *Geis. des Kindes*. Edition 1902, III, pp. 26-27. In such cases possibly each germ-plasm was normal in itself, but both did not fit well together, so that their conjugation would lead to pathological products. (This is similar to Möbius’ idea of “Keim-Feindschaft,” l.c. p. 100.)

†The taint here is usually polymorphous, not similar. In this this form differs from the other specific psychopathic constitutions (the “affective,” “hysterical,” etc.) in which the taint is so often direct and similar.

‡These *stigmata* are found singly also in normal and slightly abnormal people; but they are numerous in this type of psychopathic constitution (in about 80 per cent.). One should differentiate between stigmata of degeneracy, of syphilis and of rickets. See author’s “Hereditary Syphilis,” *Jour. A. M. A.*, April 3, 1915.

Sexual perverts, however, are not necessarily dégénérés in the above sense, nor hereditary-degenerative psychopathic constitutions. The latter state, however, is especially fit soil for sexual perversion to grow or thrive on. That the lay-term "degenerate" ought never to be applied to sexual perversion, even of the most atrocious or dangerous type, is evident, for these conditions are *pathologies* that can no more be helped by their unfortunate bearers than that of epilepsy, paranoia or typhoid. One might also keep in mind that at times abnormal sexual tendencies or perversions may be present in an individual without his personality being really modified. In such cases are not dealing with a psychopathic constitution.\*

On the ethical side we find these children lying and taking things from the earliest, and sooner or later in trouble through detection. Then the story is repeated again and again. Truthfulness and honesty appear quite impossible. At school they are truants, and hence learn little or nothing. This at times makes them appear feeble-minded. But feeble-mindedness, it should be remembered, has nothing in itself to do with the psychopathic constitution; such psychopaths may not only be intelligent, but exceptionally talented.

Liepmann cites a very interesting case, showing the possible combination of good and bad in such a character. He had under observation a psychopath who, after two previous offenses (and punishments) got away with thirteen of the most adroit burglaries possible. In his pocket was found a long poem on the death of Goethe. This poem was full of deep feeling. On the back of the sheet was a carefully compiled list of the persons from whom he was going to beg or steal, with addresses, etc. Those whom he had already taken in hand were crossed out, and the amount obtained noted.<sup>4</sup>

Feeble-mindedness *may*, however, be associated, and in fact the mildest type, *debility*, frequently is. In the latter conjunction the case is more complicated and difficult. Later these individuals become *instabiles*, constantly shifting, wandering, changing work or abode, rarely able to stick to anything or maintain themselves. From the earliest their night fugues may bring them in touch with other nightlings and bad company, and this, together with their hang to mischief, and their great suggestibility, sooner or later lets them join in the escapades of more hardened delinquents—and so the anti-social career is begun. In the case of girls, staying out at night,

\*See, for instance, K. Jasper's *Allgemeine Psychopathology*, Chap. V. 2. For general literature see Krafft-Ebing, Hirschfeld, Eulenburg, Moll, Löwenfeld, etc.

mingling with depraved company, drinking, sexual hyperesthesia, and no will whatsoever to counteract the grosser instincts, speedily leads to seduction, frequently enough to prostitution.

Mental and physical instability, squalor, drink, quarrelsomeness, temper, fits of fury, theft, prison—prison perhaps over and over again—shiftlessness, vagabondage, and the whole gamut of sexual, social and ethical depravity, this in various degrees and variegated combinations is our real *degenerate* of popular parlance.

If one studies these cases anamnesticly one will find them nearly all (probably all) hereditarily tainted, and, in fact, with severe convergent heredity, often, though not always, astonishingly consanguinous. New and good blood added to such a strain would lift it, little by little possibly clear it. A wholesome home and life would also help materially. With the children taken away at an early age, and transplanted to sunny, healthy, kindly environment, with constant sober example and uplift before them,—the curse might end, at least for many of them.\* But what good family would (matrimonially) receive such individuals, and what provision has state or society made to succor the children? Fate leaves no outlet; and so this riff-raff just hobnob with their own, mate among themselves, and inbreed their vicious traits and tendencies to a very quintessence of rottenness.

And in unrestrained fecundity they propagate, sowing with tolerable certainty the ancestral seed of decay. Their children, like themselves, grow up with vulnerable cortico-neural systems, perilously sure to prove inadequate for the daily wear and tear, reacting violently to stimulants, especially to alcohol, succumbing easily to temptation, impelled through mental and physical impotence along dubious ways of least resistance. And, as if this were not heritage

\*Gruhle's remarkably thorough study of some 105 institutional charges showed how immensely important the milieu is for the rise of ethical decay and criminality. "Die Ursachen der jugendlichen Verwahrlosung und Kriminalität. Studie zur Frage: Milieu oder Anlage." Berlin, Springer, 1912.

Meumann also wrote "Only seldom can a child before its fourteenth year develop independently of its environment into a mentally and normally self-governing individual." (See following footnote.)

Gruhle put the inherited tendencies (Anlage) at about 20½ per cent.; the milieu was the preponderant factor. An excellent example will be found in the 9th case in Pondojeff's thesis, p. 88. (See footnote 18.) Also H. Albrecht, "Die Wohnungsnot in den Grosstädten und die Mittel zu ihrer Abhilfe," 1891.



enough, think of the homes that rear their childhood, offering the vilest examples of irresoluteness, depravity, drunkenness and immorality. What other course can such creatures take but the downhill path of their fathers! Strange enough, the course is occasionally the other way—and such families, even well along the line of carious inbreeding, have normal members, upright and honest citizens of their community.

In gauging these cases and families it is very necessary to keep the several component factors in mind, and not throw the whole mess into the heredity end of the scale. What belongs there is the convergent and cumulative taint, with its especially probable and severe transmission. Secondly, there is the important factor of the home, the general milieu, the abominable example. Thirdly, there is the negative factor of lack of schooling, for such children get little chance for school, or are truants, and the school is one of the greatest means in teaching and inculcating ethical values.<sup>6</sup> For this should be remembered—it seems to be invariably forgotten in our literature—ethical ideas and values are not innate, not born with children; *they must be developed*.\*

Nor is there such a thing as a “moral center” anywhere in the brain. The thing, the attitude we term *moral*, is a highly multiplex composit, made up of many feelings (tonal feelings, to be more exact) and many mental associations. When the feelings are perverted (i.e., psychopathic tendencies), and especially when, in addition, there is a mild intellectual defect (the graver defect cases do not move at large in the community) the propensity to action that is anti-communal, anti-social—in short, criminal, is easily understood.

Not only does education in itself give strength of will, but at school, both the work, and in large measure the play among many children—team work and reciprocal obligation—teaches inhibition,† the great lever in living harmoniously with your neighbor—and the “social” point of view. Such lesson is of greatest need to these

\*Magnan, Ziehen, Höfding, Sully, etc. This will be fully brought out in a separate study on Delinquency.

†The psychopath is wont to act almost automatically, the “process of action” discharging, as it were, upon the merest whim, wish or suggestion, and this in the face of law or the other fellow’s rights and feelings.

weak-willed psychopaths, and yet they do not get the chance to imbibe it. Special out-door schools, or special teachers, might meet the problem entirely. But the school authorities say such children cannot be kept among the normal ones, and it costs money to have extra schools for the abnormal.\* In short, they have neither such money nor schools. Well, in God's name, why not then unhouse some of the normals, and let them vegetate, and turn the buildings into schools and institutions for the various psychopathic constitutions, if society (which means our legislature) has not vision enough to see farther than its nose! for, in the end, such schools and institutions will save the community hundreds of thousands in funds and an infinitude of heartache and misery, by mitigating or nullifying the vicious tendencies of these unfortunate creatures.

It is to these tendencies, as already explained, and not upon feeble-mindedness, that delinquency is due. *Feeble-mindedness has nothing in itself to do with delinquency*, though it may be a good soil (one of many soils) for it to grow on. It should be remembered, too, that psychopathic constitutions are extremely numerous, outnumbering mental defectives many times. They can be made good citizens if taken in hand early enough; the defectives are utterly hopeless. Finally, the psychopaths, if not trained and treated, are the far more dangerous of the two.

Summing up our conclusions, we must infer, then, that there is no such thing as hereditary delinquency, or a *born criminal*, no really criminal family lines, no truly criminal stock; there is only the born psychopath—either intellectually normal, or stunted by lack of schooling, or defective—the hereditarily tainted even degenerated (i.e., strongly convergently tainted) psychopathic constitution, with his uncontrollable affectivity, his lack of inhibition, his inability to weigh, reflect and judge, and withal, an inherent unrest, and a blind forward seething with so unarmored a front. Environment, seduction and the perverse vicissitudes of life achieve the rest.

But we are not permitted to decamp at this point, for a final problem is shelled out of the above, and put into our way for solution. Granted there is no born criminal, and that neither delin-

\*At present the defectives are cared for in special classes, but not the psychopaths.

quency, criminality nor the hang to wrong is heritable or transmissible, are there not individuals who are found from the very earliest to be incorrigible, unsocializable, who show no affection, and have a kind of anesthesia to feelings of sympathy, respect, thankfulness, pity, gratitude, other's pain, etc., who cannot grow into moral and ethical beings because they cannot comprehend or live up to the attitudes we term social, moral and ethical in our relations? Are these not the chronic recidivists of the courts, the high-handed egoists, uninfluencible through advice or punishment? Do they not justify our holding to the term *moral imbecile* or *moral insanity*?\* And if so, is there not a born moral defective whose problem is much the same as that of the "born criminal," and necessitates our re-overhauling the above deductions and admitting a heritable type of offender after all?

The subject will be touched on but briefly here, as I shall take it up at length in a later paper. Modern study, and especially the work of Ziehen, has endeavored to expunge this teaching, and eliminate the name "moral insanity,"<sup>7</sup> for it is an obvious misconception to think a child can have an isolated defect of ethical feeling, all his other psychic functions being normally developed—as if, as one psychiatrist put it, there could be a developmental inhibition of the "moral tract." On closer examination, after one has excluded the psychotic forms, one finds that these cases belong to one of three groups:

(1) Cases in which there is a mental debility (or defect), the ethical defect being only part of the defectivity. These cases belong under feeble-mindedness<sup>8</sup> (Ziehen's "Debilität mit vorzugsweise ethischem Defect").

(2) Cases in which there is no intelligence-defect whatsoever,

\*The term "moral insanity" was given by Prichard in 1835. It was presumed that there were individuals who, though intellectually sound, were born with moral defect, i.e., defect of the moral sphere, as it were. Lombroso's theory of the "delinquente nato" ("La pazzia morale e il delinquente nato") was published much later (Arch. di psych., Vol. III, 1882), and though it has some association with the above, is, nevertheless, very different, the criminal instincts being presumed to be atavistic. The born criminal would then be an anthropologic type—not a defective.—Nordau's theory was to the effect that criminality was due to social parasitism which developed on the soil of degeneracy (Entartung), this degeneracy consisting of a certain organic inferiority, especially a lack of inhibition impulses and a relatively rapid exhaustion of the nervous system.

the tendency to immoral action being due not to a lack of or defective ethical feeling, but to positive psychopathic trends. These cases belong to the psychopathic constitutions, especially the hysterical or degenerative. (In Ziehen's private cases they proved the majority—three-fifths belonging here).

(3) Cases in which a careful anamnesis shows that these children missed certain necessary influences which make for moral and ethical character, and were open to bad influences which spoiled them, namely, poor parental training, or lack of any parental training, the misguidance of servants, bad milieu, evil companions, even seduction, etc., etc. In such cases there is no pathological process whatsoever present but just a normal process of stunting and perverting (i.e., thwarted ethical development—the German *ethische Verkümmierung*).

Not a single unchallengeable case, writes Ziehen, have the claimants for "moral insanity" brought forth in the literature of the last forty years, not a case that could not be brought into one of these three groups.

Scholz<sup>9</sup> states the same thing a little differently—"Morally depraved we find among the instable (*Haltlosen*), the 'periodicars,' the affect-natures, as well as among the insane and feeble-minded. The problem should, therefore, not exactly be 'is there a moral insanity,' but 'can a moral inferiority also be of pathological origin?' And this second question one must absolutely affirm." The term "moral insanity" he, too, would drop, designating the *symptom*, if needs be, with "moral anesthesia."

In regard to the *psychotic* forms just alluded to, they are represented in the epileptic acting in a twilight state, the hysterical in the same condition or on perverted impulsion, the pathological intoxications, the acute rapidly passing paranoias, menstrual psychoses,\* the precoc cases, and acts associated with hallucinations, or

\*Abnormal mental states, ranging from the psychopathic to the psychotic, may occur in *normal* women during or just prior to menstruation. This may occur just once; or it may occur with each period. Such abnormal condition is more frequently noticed in psychopathically constituted individuals. It is especially apt to occur in the hysterical (always to be thought of in unmotivated shoplifting). Such menstrual incident, however, may be the prodromal to a real psychosis. For literature see Krafft-Ebing, "Psychosis Menstrualis," Stuttgart, 1902; Wolter, "Zur Lehre von den menstrualen Psychosen," diss. Kiel, 1910; P. Jolly, *Arch. f. Psycho. u. Nerven.*, Vol. 55, No. 3, 1915, etc.

such occurring on pathological impellances in cases of compulsion—(or obsession—) neuroses or psychoses. These must be clearly kept in mind if one is to probe the *pathology* behind abnormal acts and anti-social behavior, for nowhere is the necessity of refined diagnosis, of fixing our nomenclature and our symptom-complex-pictures more important than here, inasmuch as subtle similarities between psychotic and psychopathic states may exist, so much so at times as to make diagnostic differentiation impossible (especially at the time we see the delinquent—when the pathological process may not yet be fully developed).

That sufficient expert work is applied in the sifting and diagnosing of delinquency must be doubted, judging from the literature, surely not in commensuration with the importance the subject warrants; in fact as a subject per se this psychoclinicism is as yet in swaddling clothes! Everywhere one sees novices at work, and everywhere the atmosphere is clouded by Binet-testing—the trained physicians being few and far between and almost absent from the list of doughty experts. At the recent meeting of the American Psychological Association a dozen papers were presented on delinquency, all of them dealing with the Binet or modified Binet testing of these individuals, with not a thought as to pathology or diagnosis,—and yet, as has already been said, lack of intelligence has in itself nothing whatsoever to do with delinquency. The words psychopathy and psychosis were not mentioned! Diagnosis meant age-norms in terms of Binet, and nothing else. It is difficult, indeed, to understand why this science is allowed to drift so hopelessly astray—or why the medical profession does not claim this province as its own.

What unnecessary errors may occur the following case evidences: Not long ago one of my clinic patients was arrested for exhibitionism, was tried, and, though defended by a prominent counsellor, was found guilty. This young man was an outspoken psychopathic constitution, with the most evident history of epilepsy besides. That something pathological might have induced this stripping occurred to no one of the entire court, yet every neurologist is aware that such procedure is common to many epileptics as they go into attacks, or represents an attack itself.

This may not be a case upon which Binet tests would have been

made—one is not sure!—it is one though that the medical psychoclinicist should have supervised. Here is another: A young man with homosexual tendencies (now a patient of mine) wrote some letters of an erotic turn, to a younger acquaintance. These letters were intercepted, and an attempt at crime “of a foul and degenerate nature” made out against him. This poor unfortunate was sent to penal servitude, by a southern judge, for three years!

The courts ought always to keep pathological sexual impulses in mind. Sometimes they are homosexual, sometimes sadistic; but far more difficult to recognize are acts committed because of “sexual servility” to someone else, a very remarkable condition, frequent enough, yet scarcely mentioned in our literature.<sup>10</sup>

The compulsion or obsession cases are sometimes very difficult to decipher. Those that concern us here are the active type (Sommer spoke of them as “sozial störende”) in which harm to others may occur.<sup>11</sup> Sometimes through the stilling effect of alcohol or drugs, these individuals become drink or drug habitués beside. Sometimes it is scarcely possible to differentiate such an impulse from hallucination, etc. So, a boy of nine, whom I treated, stole right and left when he had the chance, and was exceptionally clever at it. His explanation was that “something in his head told him to do it” or “a voice made him do it,” etc. It was not possible, to be sure, whether there was a real voice here (hallucination) or whether this was just “metaphorically speaking” for an irresistible impulse. The boy was thoroughly neuro-psychopathic, and showed the intensest erethism.

Another patient, an epileptic, robbed two of her neighbors—and very thoroughly! She claimed amnesia for the act. From her account there was some doubt whether to believe her (would she have been believed in court?). A week later she went into the cellar during an automatic twilight state, set the place on fire and was very severely burned.

Scholz<sup>12</sup> cites on an especially interesting case—a fourteen-year-old murderess with several most horrible crimes to her name, the memory of which disturbed her not in the least, had her good parts too, namely, thankfulness, and under conditions, real warm-heartedness. She began to show periodic outbreaks of fury in prison. Two years after the murder for which she was incarcerated, she

suddenly became manifestly epileptic, had twilight states, and intensest uncontrollable excitement. She died at 23.

The cases of hysterically motivated thieving should be remembered, in spite of the public's impatience with kleptomania, and especially the cases of pathological "rage outbreaks" and crime committed during such in the affective psychopathic constitution. Finally the jealousy (or other) attacks in the paranoid psychopathic constitution (this is not paranoia, however,) seen occasionally in the alcoholics must be borne in mind.<sup>13</sup>

The problems of prophylaxis and therapeusis need not be gone into more fully in our study. The duty of the medical profession to claim this field, to further its aims and stimulate efficiency, we wish, however, emphatically to reiterate; nor will this lessen the urgency of cooperation between society, school, court and clinic, in mastering the task of wisely searching out psychopathy in our midst, of appreciating the pathologic in all abnormal behavior, delinquency and crime, and of succoring this long list of unfortunates not through punishment but through early recognition, timely training and the therapy of kindly environment and a coercing mind.

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## THE ARCHIVES OF DIAGNOSIS

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the internal periosteum of the skull, as well as the covering membrane of the brain, and as a sheath for the spinal cord. The dural sac ends abruptly at the second or third sacral vertebra.

Whereas, in the cranium the dura is closely adherent to the skull, particularly at the sutures and at the base of the skull, in the vertebral canal the dural sheath is comparatively free and unattached. Between the dura, which contains the spinal cord, and the inner wall of the vertebral canal, there is found a mesh work of connective tissue which gives support to a system of venous plexuses.

*The Choroid Plexuses.* In the fourth ventricle on each side of the midline on the under surface of the tela choroidea inferior, there are minute tufts of pia mater tissue projecting ventrally. These tufts are covered by epithelium, and are known as the choroid plexuses of the fourth ventricle.

In the third ventricle lying along the roof and projecting downward, one on each side of the middle line, are the choroid plexuses covered by specialized epithelium. On the floor of each of the lateral ventricles there is also a choroid plexus of the same histologic structure.

The epithelial cells covering the choroid plexuses are of the columnar type, and are known as the ependymal cells. These nucleated epithelial cells surmount the folds of the plexus and in some areas show ciliary processes.

In its entirety, we may look upon the interior of the brain, the ventricles of the brain, as being lined by the pia mater, which is reduplicated in folds at certain areas in each of the ventricles; these reduplications are covered by specialized cells for the purpose of a special function—secretion of the cerebrospinal fluid.

#### THE CEREBROSPINAL FLUID

*Source.* There has been but little deviation from the ideas elaborated by Quincke, that the spinal fluid is merely a secretion of the choroid plexus of the ventricles that a smaller portion of the fluid originates in the subarachnoid spaces of the brain and spinal cord, and that still another portion comes from the lymph tracts of the central nervous system which empty into the subarachnoid space.

In proof of the origin of spinal fluid from sources other than the choroid plexus, Dandy and Blackfan<sup>1</sup> cite an instance in which

they observed complete obstruction of the ventricle, in which case no fluid could have come from the choroid plexus. The fluid in the spinal canal was completely drained, and in a very short time they found that 5 c.c. of fluid had reformed evidently from the convexity of the cerebrospinal axis.

Illustrative of the part played by the choroid plexus is also the interesting experimental work of Dixon and Halliburton,<sup>2</sup> who administered intravenous injections of an extract of the choroid plexus, with an augmentation in the flow of the cerebrospinal fluid. They called the substance responsible for the stimulation of the secreting epithelium a "choroid hormone." Dixon and Halliburton, therefore, believe that there is a product of the metabolism of the brain and cord which, circulating in the spinal fluid, reaches the choroid plexus and stimulates it to secretion.

*Biologic Functions of the Spinal Fluid.* Mott and Weed<sup>3</sup> have demonstrated a system of canaliculi which surround the neurons of the cerebrospinal axis. These canaliculi are in direct communication with the subarachnoid space, and their function is to carry off the products of metabolism from the nerve tissues. From this viewpoint, Mott compares the spinal fluid to the lymph of other tissues, and holds that one function of the spinal fluid is to act as a medium of exchange, bringing nutriment to and carrying away the products of metabolism from the tissues with which it comes in contact.

Cushing and Goetch have suggested that the trace of pituitary substance found in the spinal fluid indicates that some of its constituents may be inactivators of the cerebrospinal system which incites the nervous tissues to their functions.

Dixon and Halliburton have found under certain conditions a temporary increase of carbon dioxide in the spinal fluid. They claim that the  $\text{Co}_2$  acts as a lymphagogue, increasing the flow.

Such observations as these make it appear that the cerebrospinal fluid is charged with some very important physiologic functions; and although our present day ideas of these functions are quite bizarre, yet the indications of their importance are very striking.

That the cerebrospinal fluid contains substances from other sources than the choroid plexuses is also suggested by the presence of cholin in the fluid; cholin being a disintegration product of lecithin and cephalin, the origin of which is nerve tissue. In this connection the

suggestion of Frazier seems plausible, which is that the fluid coming from the choroid plexuses and that coming from the convex surface of the cerebral hemispheres and spinal cord are of quite different chemical composition; and the spinal fluid, as we know it, is the admixture of both sources. It has also been observed that the fluid in the ventricles has a higher sugar content than the fluid in the subarachnoid space, and that the fluid in the subarachnoid space is richer in albumin than that of the ventricles.

*Physical Functions of the Spinal Fluid.* Frazier enumerates the mechanical functions of the spinal fluid as follows:

(1) as a water bed to protect the brain and cord against shock and injury; (2) to support the arteries; (3) to maintain uniform pressure; (4) to preserve uniform intra-cranial pressure during systole and diastole, during inspiration and expiration, in health and disease. Kafka<sup>4</sup> and Goldman<sup>5</sup> hold that the spinal fluid protects the nervous system in the capacity of a barrier to infection. Frazier<sup>6</sup> also has shown that the choroid plexus acts as a barrier to poisons. He injected 1 c.c. of a 1 per cent. solution of trypan blue in the jugular vein of a rabbit which stained the choroid plexus, but did no injury to the nervous system; whereas the injection of 2 c.c. of a 1 per cent. solution into the arachnoid space caused death in twelve hours.

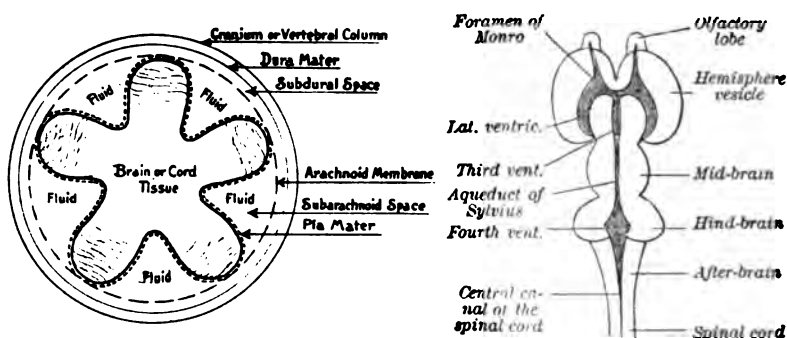
In a purely mechanical way the cerebrospinal fluid serves some very important purposes for the protection of the nerve tissues which it surrounds. The functions are, for example, ease of displacement of the fluid from points subjected to local pressure, the ability of the spinal fluid to increase or decrease in total amount to compensate for a large or small blood supply, the function of displacement which occurs with change in contour of the vertebral column, and the protective action which the spinal fluid must possess as a shock absorber.

*The Amount of Spinal Fluid.* The total amount of cerebrospinal fluid in the subarachnoid space and ventricles is estimated as being from 100 to 130 c.c. Half of the total amount is in the dural sac and from 20 c.c. to 30 c.c. in the ventricles.<sup>7</sup> This amount, it is generally held, is renewed six or seven times in twenty-four hours. Frazier gives the amount at any one time as 60 c.c. to 120 c.c., and he estimates that replacement occurs every three hours; the total

daily secretion being from 360 c.c. to 720 c.c. Cases have been recorded in which after operation or accidents, there has been a flow of 100 c.c. or more per hour, this flow continued for days and even weeks, or as long as the exit for the fluid, remained open.

*Course of the Spinal Fluid.* A large proportion of the fluid, as previously stated, is formed in the ventricles of the brain by the choroid plexuses. The course of the fluid then, is lateral ventricle, third ventricle, fourth ventricle, through the Foramen of Majendie into the subarachnoid space which surrounds the cerebrum, cerebellum and spinal cord.

What proportion of the entire amount of cerebrospinal fluid comes from other parts than the ventricles of the brain, is not explicitly known, nor as previously stated are we certain as to which constituents of the cerebrospinal fluid are contributed by the other parts than the choroid plexus.



*Factors Influencing the Secretion of the Spinal Fluid.* Frazier and Peet studied the effects of glandular extracts and other substances upon the secretion of the cerebrospinal fluid. They used spleen, kidneys, pancreas, testes, ovary, liver, adrenal, urine, bile, cerebrospinal fluid, chloroform, ether, amyl, nitrite, magnesium sulphate and physiologic salt solution. Their work showed that if a substance had any effect at all upon the secretion of cerebrospinal fluid it was to accelerate the flow. And, furthermore, they observed that as soon as the amount of the cerebrospinal fluid was increased, the blood pressure fell. Their experiments showed that thyroid extract has an inhibitory effect upon the choroid secretion and is

capable of reducing the total flow to one-third of the normal quantity. Atropin has no effect upon the secretion in dogs (Dixon and Halliburton).

*Resorption and Drainage.* There is plenty of evidence to show that absorption by the way of the venous channels of the subarachnoid space is easily accomplished. The total liquid exchange in twenty-four hours under normal conditions is, as previously stated, from 360 c.c. to 720 c.c.

Lewandowsky introduced strychnine and found it ten times as effective as when given subcutaneously. Cavazzani introduced ferrocyanide and iodine and obtained evidences of absorption and Jacobs introduced gm. 1.0 of potass iodid in a 4 per cent. solution in the spinal canal of a dog, and discovered it six hours later in the urine. It is interesting to note that Jacob in 1898 introduced 25 c.c. doses of a 0.01 per cent. solution of sodium iodid in cases of cerebrospinal syphilis and claimed to have had beneficial results.<sup>8</sup>

Frazier<sup>1</sup> has been able to introduce 500 c.c. to 1000 c.c. of normal salt solution in the subarachnoid space of a dog, in the course of an hour or two. Certain dye substances are readily absorbed from the subarachnoid space; Frazier showed that 60 per cent. of the phenolsulphonaphthalein injected into the subarachnoid space, was eliminated by the kidneys in two hours. In attempting to determine the location from which absorption took place, Frazier injected phenolsulphonaphthalein into an artificially blocked ventricle and found that no absorption took place; from which experiment it would seem that absorption takes place in the subarachnoid space surrounding the convexity of the cerebrospinal axis, rather than in the ventricles.

*Drainage.* "The outlet for the cerebrospinal fluid is by means of narrow channels through the villi of the arachnoid to the venous sinuses, thence through the subdural space and the dura to the lymph vessels of the neck and throat (possibly also the nasal mucous membrane and the labyrinth of the ear) and probably along the sheaths of the nerves of the brain and spinal cord" (Quincke).<sup>9</sup>

Mott<sup>2</sup> and Weed<sup>3</sup> hold that the larger portion of the cerebrospinal fluid drains off through the arachnoid villi and venous plexuses of the cerebrospinal cavity, while a very slow process of filtration also takes place along the lymphatic system and spinal nerve.

*Manometric Pressure.* The cerebrospinal fluid may be looked upon as a pool of liquid enclosed within the cerebrospinal cavity under a pressure proportionate to the pressure of the forces which surround it. The average normal pressure is 40 mm. to 130 mm. water, or according to some observers up to 150 mm. water.

Expressed in terms of mercury the height of the pressure is usually given at 5 mm. to 7.3 mm. Hg. The pressure is higher than the normal venous pressure and lower than the arterial. The normal pressure taken in the veins of the arm at the level of the auricle, in the horizontal position, we found in 26 young adults to vary between 35 mm. and 110 mm. water.<sup>10</sup>

While numerous observations have shown that increased pressure of the cerebrospinal fluid does not occur simultaneously with arterial hypertension, it is nevertheless true, that primarily it is the arterial pressure that is the starting point for the pressure of the cerebrospinal fluid. It is conceivable that, should the entrance of spinal fluid into the subarachnoid space be made at normal or greater than normal pressure, and absorption of the fluid interfered with, the tension of the cerebrospinal fluid would be abnormally raised.

We may well say that the cerebrospinal fluid is a pool of liquid in point of pressure occupying a position between the arterial and venous circulation; being brought there by the former and carried away by the latter.

*Physical Characteristics.* The normal spinal fluid is a limpid, translucent and transparent fluid, as clear as distilled water. It is odorless and tasteless. The normal fluid does not foam when shaken.

*Reaction.* The reaction of the fluid is alkaline and expressed in terms of sodium hydroxid, it corresponds to about 0.1 per cent. Hurwits and Tranter<sup>11</sup> studied the reaction of the spinal fluid by determining the hydrogen-ion concentration, believing that such determinations offer a more exact estimate of the true chemical reaction. Their method consisted in the addition of a solution of phenol-sulphonphthalein to the spinal fluid, and in comparing the resulting color to phosphate mixtures of known hydrogen-ion concentration. Readings were made by matching the colored spinal fluids to standardized phosphate solutions of corresponding shades.

These investigators found the average hydrogen-ion concentration was PH equal to 1.11.

*Specific Gravity.* The specific gravity of the spinal fluid is usually between 1003 and 1009. Between these points there are fractional variations in health and disease. Sanford<sup>12</sup> reports that in one form of mental disease, he finds a specific gravity of 10045, while in others he finds 10043. We will refer to this again under the abnormalities of composition.

*Chemical Composition.*

Water .....	98.74 per cent.
Solids .....	1.25 " "
Dextrose .....	0.4 to 1.0 " "
Albumin as globulin and albumose	0.03 to 0.06 " "
Potassium salts, phosphates and urea	0.15 to 0.35 " "
Fibrogen—none.	

Lactic acid, occasionally a trace. Cholesterin, a trace.

The cholin found is a distintegration product of lecithin and cephalin which are products of the brain's metabolism.<sup>13</sup> Leopold and Bernhard<sup>14</sup> give the following as their findings in ten normal children:

Total non-protein nitrogen 17 to 26 mgm per 100 c.c.

Urea nitrogen 7 to 13.5 mgm.

Creatinin 0.7 to 1.5 mgm.

Sugar content 0.07 to 0.1 per cent.

Total non-protein nitrogen averaged 75 per cent. of that in the blood.

Urea 82 per cent. of the concentration of the blood.

Creatinin 60 per cent. concentration of the blood.

There was no marked variation from the normal non-protein nitrogen in chorea, syphilis, epilepsy, idiocy and enuresis.

Mott states that the cerebrospinal fluid gives water and  $\text{Co}_2$  to the blood and takes sugar and oxygen. Under certain conditions Dixon and Halliburton have found a large amount of  $\text{Co}_2$  which they believe acts as a lymphagogue. Cushing and Goetch have found the active principle of pituitary body in the spinal fluid and they suggest that its purpose is that of an "inactivator" to the nervous system.

*Cytology of the Spinal Fluid.* The spinal fluid in the normal person gives a cell count up to about 8 cells per c.mm. These leucocytes are of the mononuclear type and their origin has been a



mooted question. Do these cells circulate in the spinal fluid as the white cells of the blood and are they a functioning part of the normal fluid or is their presence merely incidental, being cast off products from the walls of the subarachnoid space? If as Mestrezat<sup>13</sup> holds the spinal fluid is a product of dialysis; then it would seem that the cells are probably of histogenic origin or if the cerebrospinal fluid is a lymph transudate as is held by others, then the cells are probably of hematogenic origin.<sup>14</sup>

Another method of determining the origin of these cells would be to classify them according to their staining characteristics. The majority of these stained cells correspond in their morphology and affinity for dyes to the cells of the lymphocyte group, while a small portion possess the characteristics of the plasma cell, such as is found in the contiguous structures of the meningeal membrane.

*Morphology of the Cells.* The small lymphocyte is the most frequently encountered cell. It is smaller in size than the red blood corpuscle and it is poor in protoplasm and it is basophilic. The large lymphocyte is of the same type as the small one, but in size is larger than the red blood cell. The old lymphocyte is a poorly stained cell.

*The Plasma Cell* is a large cell with a deeply stained nucleus and a fine granular protoplasm.

*The Macrophage* is found in the spinal fluid as a result of irritation to the meninges; it appears in the rôle of a phagocyte. These cells are believed to be of hematogenic origin and their altered appearance and staining properties are believed to be due to their having entered a medium, the spinal fluid, which differs in chemical composition from their native medium, the blood. It is readily seen that a cell transferred to a medium of different specific gravity, chemical reaction, composition, etc., etc., would be affected in the way this cell appears to be altered.

*The Granulocyte* (cells of the polynuclear type) when found in the spinal fluid indicate a pathologic process.

*Indications for Lumbar Puncture.* As time goes on the indications for lumbar puncture are becoming more numerous. These indications may be divided into the diagnostic and therapeutic.

*Lumbar Puncture for Diagnosis.*

I. Diagnosis in the infections.

- II. Diagnosis in conditions producing pressure symptoms.
- III. Diagnosis in traumatic conditions.
- IV. Diagnosis in miscellaneous conditions.

*I. Diagnosis in the Infections.*

- A. Cerebrospinal meningitis.
- B. Pneumococcic meningitis.
- C. Acute poliomyelitis.
- D. Meningismus in the acute infectious diseases.
- E. Meningitis complicating the infectious diseases.
  - Typhoid fever.
  - Pneumonia.
  - Influenza.
  - Erysipelas.
  - Syphilis.
  - Tuberculosis.
  - Actinomycosis.
  - Trichinosis.
- F. Meningitis secondary to brain abscess, mastoiditis, sinus thrombosis.

*II. Diagnosis in Conditions Causing Pressure Symptoms.*

- A. Cerebral compression.
- B. Cord compression.
- A. Cerebral compression.
 

<ul style="list-style-type: none"> <li>(1) Diseases of the cranium.</li> <li>(2) Diseases of the membranes.</li> <li>(3) Disease of the brain.</li> <li>(4) Hydrocephalus.</li> </ul>	}	<ul style="list-style-type: none"> <li>Inflammatory reaction.</li> <li>Tumor.</li> <li>Cyst.</li> <li>Hemorrhage.</li> <li>Aneurism.</li> </ul>
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- B. Cord compression.
 

<ul style="list-style-type: none"> <li>(1) Disease of the vertebral column.</li> <li>(2) Disease of the membranes.</li> <li>(3) Disease of the spinal cord.</li> </ul>	}	<ul style="list-style-type: none"> <li>Inflammatory reaction.</li> <li>Tumor.</li> <li>Cyst.</li> <li>Varicosity of veins.</li> <li>Aneurism.</li> <li>Hemorrhage.</li> </ul>
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*III. Diagnosis in Traumatic Conditions.*

- A. Traumatism of head and neck.
- B. Traumatism of the vertebral column and spinal cord.

*IV. Diagnosis in Miscellaneous Conditions.*

- A. Unconsciousness in obscure cases.
- B. Hysteria.
- C. Epilepsy.
- D. Pernicious anemia with spinal cord symptoms.

*Lumbar Puncture for Therapeutic Purposes.* This is performed for the relief of pressure and for the introduction of medicaments.

*Relief of Pressure.*

- (1) Tumor.
- (2) Hydrocephalus.
- (3) Tuberculous meningitis.
- (4) Serous or aseptic meningitis.
- (5) Hemorrhagic internal pachymeningitis.
- (6) Choked disk and blindness of undetermined origin.
- (7) Meningismus of typhoid fever or other infections.
- (8) Chronic persistent headaches in neurasthenics.
- (9) Vertigo.
- (10) Traumatic neurosis.

*Cerebrospinal Medication.*

- (1) Cerebrospinal meningitis.
- (2) Tetanus.
- (3) Syphilis.
- (4) Chorea.

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THE INADEQUACY OF THE PRESENT CLASSIFICATION  
OF PULMONARY TUBERCULOSIS

By WILLIAM C. VOORSANGER

Medical Director, The Oaks Sanitarium  
Los Gatos, California

It must have occurred to all who are working in the realm of tuberculosis that no adequate classification of this disease has as yet been evolved out of all the accumulated ideas published during the past quarter century. Two classifications seem to stand out most prominently as having survived the test of time. These are—first: The Continental—and second: The classification of the National Tuberculosis Association. The Continental or German Classification as evolved by Turban and strongly enunciated by Von Leube and others divides tuberculosis into three stages.

It took many years to convince us that dividing tuberculosis into stages did not give an adequate idea of the patient's real condition. Then came the addition of incipient tuberculosis to these stages and later even pre-incipient. After thoroughly testing this nomenclature, however, medical opinion decreed that incipient tuberculosis as diagnosed rarely existed, that any patient showing bacilli in his sputum must have a more or less advanced lesion somewhere in either lung. The German writers added the word "Spitzen catarrh" to the three stage nomenclature with some effect, because "catarrh" or inflammation of an apex conveys the idea of a condition either tuberculous or predisposing to tuberculosis without actual destruction of lung tissue.

"Spitzen catarrh" became very popular in Germany both as a medical and lay term and was also divided into three stages: First, "Spitzen catarrh" without bacilli; second, "Spitzen catarrh" without alteration of percussion, and third, "Spitzen infiltration" without bacilli. All these divisions of "Spitzen catarrh" specify no tubercle bacilli in the sputum. The third division specifies an apical infiltration corresponding to our one time classification of incipient tuberculosis without bacilli in the sputum. The German classification, however, of "Spitzen catarrh" would have been very satisfying to cover that large series of cases without symptoms of tuberculosis, such as loss of appetite, malaise, lethargy and some loss of

weight, and a high pitched note over one apex where the diagnosis of an actual tuberculosis is in doubt. But the diagnosis of "Spitzen catarrh" became so frequent and popular that a real tuberculosis was often missed and the patient allowed to go his way in ignorance of the real and more serious condition. So Spitzen catarrh came to be synonymous with beginning tuberculosis, a situation to be deplored since today we are without a name for that condition which often precedes an active tuberculosis.

The National Tuberculosis Association in an attempt to clarify the confusion of an ever-growing nomenclature in which such terms as chronic phthisis, acute phthisis, acute miliary tuberculosis, fibroid phthisis, acute and chronic ulcerative tuberculosis, acute pneumonic tuberculosis, had crept in to complicate the three stage classification divided the disease into—First, incipient; second, moderately advanced; and third, far advanced.

Incipient tuberculosis comprises those cases "with slight infiltration limited to the apex or a small part of one lobe with slight or no constitutional symptoms. Slight or no elevation of temperature or acceleration of pulse at any time during the twenty-four hours. Expectoration usually small in amount or absent. Tubercle bacilli may be found or absent. No tuberculous complications."

Moderately advanced specifies, "No marked impairment of function either local or constitutional. Localized consolidation moderate in extent with little or no evidence of destruction of tissue or disseminated infiltration. No tuberculous complications."

Far advanced comprises, "Marked impairment of functions local and constitutional. Localized consolidation intense or disseminated areas of softening or serious tuberculous complications." (See Pulmonary Tuberculosis—Otis.)

Analysis of the National Association Classification shows that no provision is made for the passive tuberculous who may carry a quiescent lesion from his childhood infection or who has tubercle bacilli locked up in a mediastinal tuberculous gland ready at any time to break through and produce an active tuberculosis.

If we should take a large series of cases of tuberculosis carefully examined clinically and controlled by X-ray examination, we should find that most cases with active signs such as dulness, bronchial breathing and râles in one lung, have a slight lesion in the

other. Again cases without active clinical signs but with cough as a symptom have been demonstrated to have circumscribed cavities or deep central lesions.

I have three patients each weighing two hundred pounds with no symptom but cough, in excellent physical condition with "no impairment" of function; who have circumscribed cavities walled off by adhesions. One patient, a man fifty-seven years of age weighing two hundred and four pounds, was treated for three years for bronchitis. He consulted a throat specialist who finding no impairment in his pharynx or larynx to cause cough or expectoration, had an X-ray plate made and with it sent him to me for examination and subsequent treatment. This patient's status upon physical examination was absolutely negative but his sputum teemed with tubercle bacilli. Now under which of these above classifications will we place a case of tuberculosis with cavity formation, tubercle bacilli in the sputum but no impaired function? Cavity formation means advanced tuberculosis and still this man's expectation of life may be greater than the "incipient" case with slight infiltration of one apex and slight "elevation of temperature."

We all have in our minds scores of patients, who began a tuberculous career as an incipient case and was dead in six months. We have done away with "Phthisis florida" nomenclature but we still see many cases of acute progressive phthisis.

Under what classification will we put the persistent bleeder who has frequent hemoptysis without physical signs but probably an extensive hilus lesion often only discovered at autopsy?

Under what classification shall we place the patient with hidden tuberculosis who under a sheath of healthy lung tissue conceals a large softened area? This case often becomes suddenly a case of advanced tuberculosis. I have in mind two such cases. One was discharged as arrested because he showed no physical signs whatever, after eight months of treatment and all symptoms had disappeared. One week after his discharge he had a violent hemorrhage; when examination was again possible he showed all signs of an extensive cavity.

The second case—after six months of sanitarium care—was discharged as arrested having gained over twenty pounds in weight with complete disappearance of all clinical symptoms of the disease.

About one month after his discharge in apparently excellent health while walking along the street he had his first hemorrhage and dropped dead in a pool of blood. These two are unquestionably cases of deep-seated lesions which could not find a place in our present classification.

To be sure our advanced technic in röntgenology and our better interpretation of chest plates have materially helped in placing the tuberculous in their rightful classification.

The writer eleven years ago in collaboration with Dr. Lehman published the results of clinical examination of a series of tuberculous cases compared with X-ray findings, demonstrating that "the X-ray affords valuable information as to the extent of the lesions which were often more widespread than the ordinary clinical evidences seemed to suggest." (*Am. Jour. Med. Scie.*, Oct., 1906.) This statement made eleven years ago has been proven by many observers since, although there are still numerous able medical men who do not believe that the X-ray is a material aid in the diagnosis of pulmonary tuberculosis.

If more careful clinical examinations were made with X-ray corroboration in practically every case, tuberculosis would not only be earlier but better diagnosed; classification would become more exact and prognosis, which should be the real purpose of any classification, could be defined with some degree of accuracy and certainty.

Classifications up to the present time have been unsatisfactory because they have been based either upon pathology or upon etiology. We have never tried to classify tuberculosis absolutely clinically.

Fishberg in his recent book states, "Laennec showed clearly the unity of the elemental pathological changes found in phthisis, and Koch discovering the tubercle bacillus, proved it etiologically. But all attempts to impose this unity on the clinical manifestation of tuberculous disease of the lungs have failed dismally. In pathology, particularly in clinical medicine, unity of causation does not always indicate unity of effect. Especially is this true of a polymorphous disease as pulmonary phthisis." Again, to quote from the same author: "If the object of this classification (referring to the National) is to define prognosis of phthisis, it fails utterly."

I am firmly convinced that no adequate classification of pulmon-

ary tuberculosis can be conceived until we realize that the clinical manifestations of the disease such as toxemia, loss of weight, temperature and loss of appetite must be our basis for the stage of the disease with the presence of tubercle bacilli in the sputum or the necrosis of a certain area of lung tissue or even cavity formation, as important but secondary features. A small active lesion in one lung coming under the present classification of incipient might be very virulent, causing intense toxemia and extreme loss of weight. Whereas a very advanced lesion in both lungs might be quiescent as in fibroid phthisis and the patient live for many years. We no longer classify our cases therapeutically under a pathological but under a clinical heading.

We speak of clinical arrest—A pathological arrest of tuberculosis is achieved only in rare instances. In fact, we are only interested in curing clinical manifestations and returning the patient to a state of well-being and his former place in his world of activity. We can never guarantee that all lesions in his lungs are healed. We have learned that the prognosis and arrest of pulmonary tuberculosis does not depend upon the extent of lesions in the lungs nor upon the presence of tubercle bacilli in the sputum, but upon the natural resistance. Our only hope for tuberculin today lies in its possible organism lacks natural resistance; the advanced case becomes clinically arrested because the organism possesses this valuable essential.

All our treatment today is based upon the stimulation of natural resistance. Our only hope for tuberculosis today lies in its possible ability in certain cases to increase the activity of antibodies already in the organism. We really add nothing when we administer it. Why then should classification or diagnosis, if you like, still remain pathological? Why not clinical?

There have been some attempts to classify phthisis pulmonalis clinically. Pottenger (Jour. A.M.A., Jan. 8, 1916) classifies the symptoms and physical signs of the disease under these headings: First, those due to toxemia; second, those due to reflex action; third, those due to tuberculous process per se.

Burmand of Leysen states, "that the course of the pulmonary trouble in patients often belies the diagnosis." Head of Minneapolis speaks of "persons showing symptoms of nervous and physical ex-



haustion (toxic symptoms)" with clinical signs of tuberculosis and could be considered quiescently tuberculous. Such cases certainly exist and could find no place in our present classification. There is also a very considerable number of cases of pulmonary tuberculosis which although pathologically advanced are clinically passive. As a type of this class I cite the case of a woman of forty-two in excellent physical condition who consulted me for a "dry" cough which occurred principally during the night and disturbed her sleep. The cough had persisted for a year with no other symptoms of tuberculosis. Her weight was constant. Sputum examination revealed the presence of tubercle bacilli in as great numbers as I have ever witnessed. Persistent treatment for a year ameliorated the cough somewhat, but had no effect upon the presence of the bacilli. It is now four years since the original examination of the sputum, which still teems with bacilli, although the patient is in the best of health and clinically well. X-ray examination showed extensive involvement of both lungs, which clinically could not be detected. Pathologically this case is actively tuberculous; clinically it is passive.

The present classification being mainly pathological depends upon individual interpretation as to the amount of involvement; a clinical classification would remove this personal equation. We should attempt to classify pulmonary tuberculosis as we find it based primarily upon its symptoms and prognosis or expectancy of life. The latter is not dependent upon changes in auscultation or percussion. We have all seen crepitant râles over an entire lobe disappear after two weeks' absolute rest in bed. But we have also often seen a patient lose weight week after week with continuous fever baffling all treatment and practically no physical signs. It would, therefore, seem desirable when diagnosing tuberculosis to consider first: Is it passive or is it active? Second: Is there extreme toxemia and can this be conquered? Third: Has the patient sufficient natural resistance to offset the effect of his own inoculation?

For years I have kept the above three tenets in mind, whenever examining a new patient and using them as a basis for prognosis and subsequent conduct of the case. The term advanced tuberculosis has such terror for the sufferer from phthisis that it should only be reserved for the absolutely hopeless patient, not for the one

who shows extensive involvement upon physical examination. It would seem, therefore, that tuberculosis could be clinically classified under these very simple headings:

1. Passive tuberculosis.
2. Active tuberculosis.
3. Advanced tuberculosis.

Realizing the complex nature of a consuming and destructive disease, such as pulmonary phthisis, its manifold clinical manifestations and its often deceptive and treacherous course, no classification can encompass all cases. The above classification, however, is based upon the actual clinical condition of the patient; his ability to get well or not; and his expectancy of life, disregarding the amount of infiltration which may be present in his lungs.

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## A DISCUSSION OF THE MECHANISM OF MENTAL TORTICOLLIS

By L. PIERCE CLARK

New York

Probably no functional nervous disorder has a worse prognosis than mental torticollis. Many cases improve for a time under treatment. They may even have short periods of remission and then the disorder may recur and ultimately defy any and all systems of treatment.

The general mental makeup has largely been held to be the primary fault in the torticollitic. It has been stated that the essential constitutional defect is one of emotional infantilism. In this term is embraced those defective instincts which make the individual possessing them an indecisive, vacillating and erratically unstable person. In a careful analysis of several such persons this view seems to be sustained. All of the individuals studied showed abundant infantile reactions which were handled fairly well until the extra pressure of some new adult adaptation was sufficient to bring about the torticollis. The primary fault in character differed from that seen in the ordinary neurotic inasmuch as when the fault was brought to light the defect did not vanish, but new

attitudes had to be assumed by these persons. They were obliged to re-educate themselves, improve these weak trends of the instincts. It was as though their emotional life had been arrested or greatly retarded, and the defects brought out in the analysis had to be lived out or be made more adult. Psychoanalytically one would say these persons had suffered an emotional fixation in the infantile life. Those who could be analyzed, in part at least, did not show these so-called fixations so sharply as one sees them in other neurotics. It seemed to be a diffused weakness of the emotional trend somewhat comparable to that seen in dementia precox cases who cannot make the adult emotional life. The feelings of inferiority and inadequacy were like those which one sees in profound psychasthenics. This being the case one can understand why such persons do not readily submit to ordinary psychoanalysis. One breaks down the morbid mechanism of their neurotic disorder, but in doing so by the ordinary technic one also destroys the framework of morbid symptoms—the crutches, as it were, by which they get about, though with such a halting and limping gait. The profound degree of transference necessary to get at these individuals at all shows how marked the infantile emotional pattern is in the torticollitic. Weeks and months of readjustment of the emotional life by re-education must be instituted between certain phases of analysis. If one is not willing to part company with the strictly technical psychoanalytic method in handling torticollitics, then this line of treatment will be of little avail. By the latter aids I believe the great majority of cases can be cured. Four out of five of my cases are completely well. It takes, however, an enormous amount of time and patience to cure this disorder. But I believe my experience has laid the way open to what can be done if we will.

As regards the particular type of muscular movement in torticollitics: In all cases I found, first of all, that the torticollis was a defense mechanism—a turning away from an adult adaptation; but further analysis showed that the type of movement was even more dynamic than a regressive one alone. It was a reversion or regression to a type of movement that had the deepest pleasurable content in the infantile life. In psychoanalytic phraseology, they all were *muscularly autoerotic*, and had placed great dependence upon these satisfactions in infancy.

In brief, then, the torticollitic is a profoundly neurotic individual whose infantile emotional life is an arrest or fixation in a diffused way on the parent, on himself, and to a less degree upon his own sex, and because of such an emotional arrest his main trends of character are weak and inadequate. In early adult life with new adaptations to the complicated life tasks in his work, and especially toward the opposite sex, he breaks down with pronounced and outspoken psychoneurotic symptoms. Usually the torticollis is not a real part of the nervous symptoms as they first appear, but occur later with the more constructive efforts at repair of the nervous illness; it is then a defense or a turning away from the intolerable difficulties of life and a regression to certain crude acts or movements with an intense satisfaction content to the unconscious, and therefore infantile, life.

Though we now have a fair knowledge of the makeup of the torticollitic and a well-detailed pattern of his infantile emotional life, as well as an interpretation of the use or misuse of the torticollitic movement, we do not as yet know why this particular type of individual uses a torticollis rather than any other regression and infantile mechanism. Even the union of a psychologic and a physiologic interpretation does not yet make this definite and clear.

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## DIAGNOSIS AND PROGNOSIS OF URINARY STONE IN THE INFANT AND CHILD

BY ARTHUR N. COLLINS

Duluth, Minn.

An adult may refuse for a time to pay heed to the pain or other disquieting signs of urinary calculus, but the infant or child will usually respond to pain or "hurts" by crying. If we are alert, therefore, we shall follow up these warnings to an early diagnosis. If, on the other hand, the process of lithogenesis is insidious and pain is not the initial symptom, our analysis must proceed along lines which may severely tax our diagnostic acumen.

## DIAGNOSIS

Urinary stones seem to be found more commonly in the following locations:

1. The kidney pelvis.
2. Junction renal pelvis and ureter.
3. Abdominal portion of the ureter.
4. Pelvic portion of the ureter.
5. Bladder.
6. Urethra—
  - (a) fossa navicularis
  - (b) bulbous portion
  - (c) prostatic urethra
  - (in order of frequency).

An analysis of the urine cannot always be relied on to reveal the presence or location of urinary stone, though the urine findings are often suggestive. Microscopic examination of the sediment in a suspected case may disclose blood or pus cells, or crystals of oxalate, phosphate or of uric acid. The urinalysis, however, presents certain constant characters in that the urine may be somewhat abundant, the specific gravity 1.023, 1.025 or 1.030, the acidity often considerable and a fine precipitate of flakes of uric acid crystals, brilliant and orange colored, may be found, the uric acid crystals predominating.

Taylor, quoting from Cabot's statistics, states that in renal calculus cases:

The urine was normal in.....	14 per cent.
Albumin occurred (varying amounts).....	74 per cent.
Blood .....	68 per cent.
Pus .....	68 per cent.

Hewes believes any renal calculus will betray itself at some stage by abnormal urinary signs.

The history of elimination of sand is an important matter to determine, if possible; also, the history of frequent or painful micturition should be recognized as suspicious of stone. Hematuria accompanies nephritic colic, especially with oxalic gravel, less frequently in the interval between crises of colic. Hematuria of bladder origin is thought by Mosseaux to be very rare in the infant.

Palpation of the kidney, ureter and bladder regions may not

reveal the site of the trouble until late in the attack. If negative at first, it should, therefore, be repeatedly tried. Eliot reports a case in which the kidney was very much hypertrophied, but was misplaced by an accumulation of gravel (presumably in the kidney pelvis). Murphy's "fist percussion" in a modified form may be used over the kidney regions at the back by employing instead of the fist as a pleximeter, fairly vigorous percussion with one or two fingers of the right (or striking) hand. This method may serve to differentiate between involvement of the right or left kidney early in the attack.

Differentiation must at times be made from affections of the liver (rare in infants); also lumbago (rare), Pott's disease and appendicitis. In a case of Weill's the latter was clearly to be considered in the diagnosis. Nephritis is not rare in the infant. Casts should be searched for. Renal tuberculosis in the infant generally runs an acute course and other organs are involved. Cancer in infants or young children is not accompanied by renal or ureteral pain. Renal tumefaction should be sought and an examination made for symptomatic varicocle, "always pathognomonic to that age," according to Charvin. In infected calculus, pus and very little blood may overshadow symptoms of its presence.

Abdominal pain of a spasmodic character accompanied by vomiting, but without diarrhea, the maximum pain traceable to the kidney or along the ureter, dysuria, rapid recovery from the attack and that followed by passing gravel, leaves little else to complete the diagnosis.

In the diagnosis of stone in the bladder a metal sound may easily be passed in the female, less easily in the male, or a small cystoscope may be used, depending upon the age of the child. Mertz reports having done successive litholapaxies for bladder stone in a child of three years, in addition to several cystoscopic examinations following the litholapaxy.

Rectal examination may be useful in the diagnosis of bladder stone, also of ureteral stone. Fagge and Robinson are each quoted by Rafin as having made diagnosis of calculus in the ureter by this method.

At first the child may cry a little on urinating when no external evidences of irritation are visible. Frequency of urination may

play a part here also. As the trouble progresses the bladder pain becomes evident, when the child is active. There is greater pain on urination, leading in some cases to tenesmus and rectal prolapse. Enuresis, accompanied by night crying, occurs; sudden cessation of urine flow may also be a common symptom in some cases. (Mertz and Williamson.)

Ureteral catheterization in the infant is practically impossible. It is difficult to say at what age such instrumental procedures begin to be practicable. Rafin separated the urine in a little girl of six months.

If the radiograph is important as an agent in diagnosis on the adult, it is of even greater importance in the diagnostic endeavor on the infant. Here it has served to point out many errors which occurred in diagnosis before, and it should be advised in all suspected cases. Hartman speaks of a case in which a radiograph was not made and the surprise was great at finding stone at autopsy.

In our day diagnosis should not be considered complete without having a good, clear radiograph. It may not always be complete with the radiograph, but it is certainly not complete without. Radiographers believe that a positive plate is more dependable, however, than a negative one, although there is a small degree of error in both negative and positive. In 93 of Leonard's cases 5 failed to find stones. In 233 of his cases with negative diagnoses a stone was found at operation, or passed, in 4. On the other hand, Beneke (1914) examined 2,400 cases and found no bladder stones. Carl Beck advises radiographing the kidney region in addition to the bladder region when vesical calculus is suspected. It is very desirable that a radiograph be obtained which shows clearly all kidney, ureteral and bladder regions, i. e., the total urinary system. In infants and young children two plates should be sufficient, and usually one large plate will suffice. When two plates are used one should show both kidney and upper ureteral regions; the other should include the bladder and lower ureteral regions. Bladder calculi will be found more often if a systematic radiograph of all cystitis cases is carried out. The necessity of an instantaneous exposure in the case of a crying and wriggling infant needs no elaboration. Immobility of respiration is practically impossible.

The dorsal position is found to be the best. The thighs should be flexed, even to a right angle if necessary, in order to eliminate the "saddle-back" and to permit contact of the back with the plate. A purge is advisable before the radiograph is made. The table should be warm to lessen the trembling.

Urethral stone impacted may be ushered in by bloody urine—the child screaming with pain. Sometimes convulsions occur. The attack may be preceded for a time by difficult urination, or dribbling, or other described symptoms of bladder stone. The child is apt to be nervous or apprehensive.

The knowledge of the localization of stones simultaneously in different parts of the urinary tract is very important. A direct relationship exists between the multiplicity of localization and gravity of operation. Multiplicity of localization also darkens the prognosis.

#### PROGNOSIS

Charvin states that lithiasis in the adult may be traced back to infancy. This would lead one to believe that the immediate prognosis is good. Gravel, it is true, is generally of long duration (see Table 3). Walther quotes Geraghty that 75 per cent. of stones which enter the ureter are spontaneously expelled. He also observes that 95 per cent. of cases are unilateral.

TABLE 3.—TO SHOW IN THE CASES OF CHAVIN, MONSSEAUX AND PORTER THE INTERVAL OF TIME BETWEEN THE FIRST ONSET AND TIME OF ADMISSION; ALSO WHERE STATED, SIZE OF STONE RECOVERED.

<i>Case</i>	<i>Age on Admis'n Years</i>	<i>Age at Onset Years</i>	<i>Interval Years</i>	<i>Remarks</i>
<b>Charvin</b>				
4	3½	?	?	Autopsy: Right kidney crammed with small stones. Largest were 12 x 8 mm. (½ x 3-10".)
9	10	8	2	Urate calculi.
11	15	14	1	Stone size small pea.
12	12	9	3	Stone wt. 8 gm
13	6	3	3	



<i>Case</i>	<i>Age on Admis'n Years</i>	<i>Age at Onset Years</i>	<i>Interval Years</i>	<i>Remarks</i>
16	10	?	..	$\frac{3}{4}$ x $\frac{1}{4}$ "; cylindrical.
17	9	4	5	
18	6	$\frac{3}{4}$	5	Stone 8 x 6 x 4 cm. in size
19	11	9	2	
20	10	2	8	
21	12	11-12	$\frac{1}{2}$	
22	5	$4\frac{1}{2}$	$\frac{1}{2}$	Stone wt. 33 gm. Stone wt. 18 gm.
29	7	4	3	
32	14	12	2	
35	4	4	0	
37	15	15	0	
39	4	3	1	
40	7	?	?	
41	$11\frac{1}{2}$	1	10	
42	$11\frac{1}{2}$	3	8	
43	9	6	3	

**Adult Cases**

46	16	10	6	Stone wt. 25 gm.
47	18	12	6	
48	19	15	4	
49	19	7	12	
50	20	12	8	
51	24	4	20	
52	24	6	18	
53	46	5	41	

**Monsseaux Cases**

1	19	5	14
2	21	12	9
3	31	10	21
4	33	15	18
5	24	12	12
6	29	7	22
7	43	8	35
8	48	13	35

**Porter Cases**

1	4	$\frac{1}{2}$	$3\frac{1}{2}$	Stone wt. 3.9 gm. Size $\frac{7}{8}$ x $\frac{3}{8}$ ".
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Average 9 years. Longest 41 years. Shortest 6 months or less.

In Table 3 it will be seen that the interval of time between the date of onset, or at which symptoms pointing to disturbances in the urinary tract occurred, and the date of admission, or such time as the patient presented himself for treatment, varies within wide limits. In the 38 cases tabulated it will be seen that the interval above mentioned ranged from 6 months or less up to 41 years, the average being 9.7 years. This is in evidence of the tendency to chronicity with respect to urinary stone. And here we return to the principle of "survival of the fittest." A child well blessed with physical vigor and development will survive the presence of a calculus in his urinary tract longer than one constantly or intermittently ill with intercurrent disease.

Stone in the female bladder if not passed spontaneously may be removed via the urethra. If allowed to remain, in other words, all the symptoms common to those in the adult may develop. After removal, recurrence is possible so long as infection persists. Litholapaxy offers a better prognosis in properly selected cases than the more formidable supra-pubic operation. Convalescence after this process occurs in a brief period of hours, as a rule, and the parents are not so distraught.

Renal operations assume a different aspect. While the infant resistance to this major proceeding is small compared with that of the adult, there are numerous and increasing reports of successfully operated cases. On the other hand, Cabot and Crabtree maintain that the danger of progressive destruction of the kidney if the stone remains is larger. H. Joseph has made a very praiseworthy study of 40 cases *post-mortem*, in which stones were found in the kidneys or elsewhere in the urinary tract. None of these were diagnosed during life. Of this material 65 per cent. were bilateral cases. I have arranged some of his statistics in tabular form, as will be shown in Table 4.

TABLE 4.—COMPILED FROM JOSEPH'S FORTY AUTOPSY CASES IN INFANTS TWO YEARS OR UNDER, TO SHOW THE RELATIVE FREQUENCY WITH WHICH URINARY STONE AND COMPLICATING HYDRONEPHROSIS ARE ASSOCIATED WITH INTERCURRENT DISEASES IN OTHER REGIONS OF THE BODY.

Post-Mortem Findings.	<i>Nephrolithiasis</i>			<i>Hydronephrosis</i>				Total.
	Left Kidney.	Right Kidney.	Bilat.	Left.	Right.	Bilat.	Bladder Stone.	
Atrophy .....	1	2	2	1	..	..	..	6
Tracheitis .....	..	..	1	..	..	..	..	1
Enteritis .....	2	4	8	2	1	..	..	17
Otitis .....	1	1	2	..	..	..	2	6
Gastro-enteritis .....	..	..	2	..	..	..	..	2
Abscess submax gland.....	..	..	1	1	..	..	..	2
Cardiac dilatation .....	1	1	4	1	..	..	..	7
Bronchitis .....	1	..	1	1	..	..	..	3
Edema pia mater .....	..	..	1	..	..	..	..	1
Hyper. mes. lymph-nodes.....	..	1	3	..	..	1	..	5
Rachitis .....	..	2	4	..	..	..	..	6
Hemorr. pia mater.....	..	..	1	..	..	..	..	1
Hydrothorax .....	..	1	..	..	1	..	..	2
Lobar pneumonia .....	..	2	5	1	1	..	..	9
Ascites .....	..	1	..	..	1	..	..	2
Broncho-pneumonia .....	1	2	7	..	..	1	..	11
Meningitis .....	..	..	2	..	..	..	..	2
Hydrocephalus .....	..	2	2	1	..	..	..	5
Colitis .....	..	1	2	1	..	..	..	4
Cystitis .....	..	..	1	..	..	..	..	1
Abscess mult. cutaneous.....	..	..	1	..	..	..	..	1
Osteochondritis .....	..	..	1	..	..	..	..	1
Miliary tuberculosis .....	..	..	1	..	..	..	..	1
Intestinal tuberculosis .....	..	..	1	..	..	..	..	1
Pulmonary tuberculosis .....	1	..	1	..	..	..	1	3
Meningeal hemorrhage .....	..	1	1	..	..	..	..	2
Diphtheria .....	..	1	1	..	..	..	..	2
Carbuncle & furunculosis.....	1	..	1	..	..	..	..	2
Pararectal abscess .....	..	..	1	..	..	..	..	1
Totals.....	9	22	58	9	4	2	3	107

It will be seen from this table that diseases of an infectious nature occur in association with calculus and hydronephrosis 70 times out of 107, or 65 per cent. Enteritis and gastro-enteritis are associated with urinary stone and hydronephrosis 19 times; rachitis 6 times, and lobar and broncho-pneumonia 20 times.

Diseases of respiratory tract associated with stone or hydronephrosis 29 times.

Diseases of alimentary tract associated with stone or hydronephrosis 25 times.

Systemic diseases associated with stone or hydronephrosis 15 times.

Nervous system associated with stone or hydronephrosis 11 times.

Glandular system associated with stone or hydronephrosis 7 times.

From the above tabulation it appears that while calculus occurred more often in the right kidney, yet hydronephrosis was more frequent in the left. Stone and hydronephrosis occurred in the left kidney an equal number of times. In the right kidney stone occurred 22 times and hydronephrosis 4 times. It would therefore appear from this that the patient stands an even chance of hydronephrosis if the stone occurs in the left kidney, whereas the patient stands only 4 chances to 22 (or 2 to 11) of hydronephrosis if the stone occurs in the right kidney.

The evident predominance of bilateral involvement, as shown by Table 4, bears out Rafin and Charvin in that the prognosis becomes more somber with multiplicity of involvement. On 76 observations at autopsy Legeu found 36 (nearly 50 per cent.) cases of bilateral calculi. Kuster, who bases his study on clinical facts, arrives at the proportion of 11.78 per cent. bilateral stones. Leroy found 22 per cent. of bilateral renal or ureteral stones. Of the cases reported on by Rafin he found 4 bilateral cases, or 8.8 per cent. Bland-Sutton (1916) found that in 10 per cent. of his lithiasis patients stones were present in both kidneys. Watson (1913) has placed bilateral involvement at 30 per cent.—but not at the same time. In 19 per cent. stones were present on both sides at the same time.

A highly important lesson to be drawn from the preceding information, therefore, considering the high mortality of cases of

multiple involvement, is that the cases must be discovered and treated earlier, before destruction of kidney substance progresses to cripple the organ, or infection becomes a complication. Infection is the *bête noir* of the clinician treating lithiasis patients.

Cuturi (1911) determined from his animal experiments three very potent conclusions: (1) *B. coli* reaching the healthy kidney by way of the blood stream causes no lesion. It simply causes bacteriuria. (2) *B. coli* in the kidney complicated by calculus provokes pyelonephritis and pyonephrosis. (3) Experimentally, by separation or ligation of the rectum for 24 hours one obtains a pyelonephritis by the passage of *B. coli* to the kidney by way of the blood, after ligation of the ureter. If, therefore, the above conclusions apply to the human, an individual harboring a primary stone runs a greater chance of secondary infection when constipation exists.

When the kidney has battled valiantly without surgical relief to rid itself of the demoniacal stone within its vitals and has ceased the struggle, there occurs a complete disappearance of symptoms; but the relief affords a sense of false security, for in the words of Rafin: "*Ce silence sera le signe de la Morte de l'organe*"—the silence will be the sign of the death of the organ.

Ureteral stone as well may occasionally take on a serious aspect if it becomes incarcerated. Guisy reports the case of a little girl of 12 in whom a mixed urate and phosphate stone became lodged in the ureter and caused a lumbar abscess, necessitating incision and removal of the stone. Where bilateral involvement occurs the prognosis is brighter where the patient is able to stand operations on both sides at one time. If but one side is treated there is grave danger of anuria, presumably from reflex inhibition of renal function enhanced by the presence of stone. Gibbon (1913) operated on 4 cases with bilateral involvement by a unilateral operation. All four developed anuria in 10 to 23 days. Two of these were re-operated at the onset of anuria and recovered. The other two were not operated on and died.

A large number of Monsseaux's patients, many of them older children, were subjects of migraine headache, eczema, epistaxis and conjunctivitis; some were asthmatic, some had urticaria at intervals, others had transient articular manifestations without fever or swell-

ing, but some pain. In his opinion, if the infant is without hereditary taint, the calculus of an accidental sort due to alimentary disorders, poor hygiene, etc., the prognosis is not too severe and the patient's chances are good under better conditions. If, on the contrary, the infant is an inveterate arthritic subject, with bad heredity, presenting at the same time habitual manifestations of the diathesis, is subject to migraines, epistaxis, gastrointestinal trouble or joint-pains, tends to obesity—in a word, is not uremic or arthritic on occasion but by temperament, and is rebellious to treatment and subject to recurrences, the outlook is not so good.

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## CARDIOSPASM IN INFANCY

By LE GRAND KERR

Visiting Pediatricist to the Methodist Hospital; Consulting Pediatricist to the Williamsburgh, Bushwick, Swedish, Caledonian and Rockaway Beach Hospitals, the Industrial Home for Children and Society for Prevention of Cruelty to Children (Brooklyn).  
Brooklyn-New York

Mary M. (who I use as a type) was seven and one-half months of age when I first observed her. She was born at full term after

a normal labor and weighed seven pounds. She was breast-fed for four weeks when a deficiency in the supply made recourse to mixed feeding desirable. This was continued for another three weeks when modified milk was wholly substituted for the breast feedings. Things progressed well for three weeks when she suddenly experienced a short, sharp attack of diarrhea accompanied with vomiting but which yielded readily to a cereal water diet. Within four days she was back on her usual formula. At five months she weighed fifteen pounds and was experiencing a happy, normal development.

At six months she suffered from what was diagnosed as influenza and lost some weight and was left quite perceptibly depressed.

For the past four weeks she has shown an increasing aversion for her food. The mother states that "while the baby seems very eager for the bottle and goes at it ravenously, she quickly pushes the nipple from her mouth with a sharp cry and brings up a mouthful of the milk apparently just as it went down. This is repeated at some feedings as often as nine or ten times and then the baby will finally drain the bottle, appear satisfied and go to sleep. But at some feedings the cry and the eructation is repeated without any let-up and the baby will take none of that feeding."

During the past month, the composition of the formula has been changed fourteen times and the condition has been getting worse.

The physical examination shows a fairly developed and nourished baby with moderate pallor and a somewhat flabby musculature. The fontanel is slightly depressed. The heart and lungs are normal. The spleen is not palpable. With the exception of a slightly distended abdomen the examination shows nothing else abnormal; there are none of the bony changes of rickets and the temperature is normal.

The negative physical examination plus a normal temperature rule out causes outside of the digestive tract and militate against the probability of a bacterial infection. It is reasonable therefore to look for the trouble in the food and this has been done as is evidenced by the frequent changes to find a food that agrees.

This history is quite typical of a group of seven cases observed in the past several months the only difference in the cases being those of minor details.

John S., eleven months old, a full term infant with a normal birth weighed eight pounds. He was started on a weak mixture of cow's milk with the subsequent history that his gain was rather slow until the third month. The formula was then strengthened by adding cream and the gain in weight was more noticeable. There was never any marked disturbance in the digestion unless the infant was forced in his feedings and then there would be some vomiting. The baby always was more or less colicky and passed considerable gas from the bowels. There was never anything seriously wrong with the infant although the parents noted that he was not quite as active as other babies of his age.

At seven months the infant became very restless and apparently refused his food. He would be eager for it, but would refuse to take it after the first swallow or two. He would apparently have considerable pain at this time and it was suspected that he was teething. He began to vomit small quantities of the milk as soon as it was taken and so the formula was changed several times but without apparent relief. This attack of distress would continue for one, two or three days and then suddenly disappear and it would be supposed that finally the right food had been found. But within thirty-six hours, the same performance would be repeated.

The physical examination revealed a small, thin, moderately pale baby with flabby musculature. There were bony evidences of rickets, a much enlarged and lax abdomen, an enlarged liver and a widely open, but flat fontanel. Otherwise the examination is negative.

From the physical examination we can hardly justify any other diagnosis than malnutrition and a considerably well developed rickets without important deformity.

In seeking the cause of the malnutrition in the history, the factor of slow gain in weight, the absence of any marked symptoms of indigestion over a long period with the one exception of colic, reasonably suggest a too high fat and this is substantiated by the persistence of small, crumbly, light-colored stools.

But the history from the seventh month on, shows nothing definite, and the several changes in the composition of the food brought no continued relief. The diagnosis of intestinal indigestion aggravated and prolonged by rickets seemed reasonable.



This history is typical, except in details, of another group of four infants.

These two histories are representative of two groups of cases of infants suffering from what might readily and reasonably be diagnosed as faulty feeding. The eleven cases as represented by both groups are similar in that they are all artificially fed babies and all are more or less malnourished. In the first group of seven, however, there was the occurrence of intercurrent disease a short time preceding the apparent aggravation of symptoms attributed to the nutritional fault. Influenza was the factor in two instances, bronchitis in two, pneumonia in one, measles in one and acute gastroenteritis in one. But in none of these was the malnutrition marked.

In the second group, there was no intercurrent disease but besides being malnourished, each of the four gave considerable evidence of rickets.

In the eleven cases there were these outstanding facts:

1. There was the occurrence of the vomiting of unchanged milk in very small amounts.
2. The vomiting was always preceded immediately by a sudden, sharp cry, or other unmistakable evidences of distress.
3. The act was not associated with any change in the diet.
4. There was no apparent change in the physical condition of the infant for a considerable time preceding the occurrence of the act.

These facts taken singly might mean anything but taken in association with each other and with all the other facts in the cases, they suggest their independence and disassociation with the composition of the food.

In elucidating the history of a sudden aversion to food as given by the mothers it was clearly established in each instance that what was an apparent aversion did not exist in fact. Instead, there was a true inability to partake of food in the usual, normal manner.

This was further emphasized by the fact that in some instances after a period of persistence, the baby was able to retain the food and did so without any further evidences of discomfort or further interference with the nutrition.

There was noted in all of the cases a peculiar inconstancy in re-

gard to the persistence of the inability to retain the food and this varied not alone on different days and on different foods, but also at different nursings on the same day and upon a similar food.

It was these considerations that led me in the first instance to use the only certain means of recognition of the real cause of this peculiar type of vomiting with discomfort, finding with my use of the stomach tube that I was dealing with cardiospasm. As soon as the tube reached the end of the esophagus, its further progress was arrested at the cardia and it was usually several seconds and in one instance over a full minute before any progress could be made.

It was very evident that this obstruction was brought about by the local irritation of the tube or the food but I am not certain as to the exact cause which must underlie this local manifestation of spasm.

It was noted that extremes of temperature of the food excite the occurrence of spasm.

The power to contract resides in the cardia itself but the power of relaxation is controlled from the medulla and through the pneumogastric, inhibitory impulses proceed to the cardia. During each act of swallowing, these inhibitory impulses pass, resulting in a relaxation of the cardia to receive the bolus of food. It is easy to discern what happens if this inhibitory control is interfered with so that there is partial or complete failure to relax.

The food being unable to pass quickly into the stomach and therefor acting as a foreign body probably aggravates the failure of the cardia to relax into a condition of actual spasm of either the cardia or the esophagus, or both, with resultant discomfort and expulsion of the food.

Spasmophilia or the spasmophilic diathesis may have some influence, how much, if any, I am not prepared to say. As the spasmophilic diathesis is supposed to be more or less hereditary and shows its most marked manifestations between six months and two years and during the winter months, its influence has seemed to me, doubtful.

There is this, however, to say about the spasmophilic diathesis, that it is practically certain that it is dependent upon a disturbance in the metabolism of calcium and this in turn may be due to parathyroid inadequacy.

As these babies were receiving a sufficient amount of cow's milk to amply supply the requisite amount of calcium, there was no apparent lack in that particular although we must always remember that the calcium in cow's milk, like its other ingredients, may not be as readily taken up as in breast milk.

In the treatment of this distressing condition, I have experienced uniformly good results. Within a very few days (and at times, almost immediately) the evidences of spasm are markedly relieved or entirely disappear. It is an easy matter, however, to excite them again by any relaxation of the treatment within a period of two weeks. This is probably due in part to the influence of habit as it applies to neurotic infants in particular.

The first consideration in successful treatment is to secure the adequate rest for the infant. For a period of two weeks, the baby must be kept in a quiet, well ventilated room, with one attendant, no visitors and no family. Usually because of their recent discomfort these infants have been coddled, handled, excited by attempts at amusement and otherwise maltreated. A room at the top of the house capable of proper ventilation is best and the furnishings should be such that quiet may be maintained. I always have the legs of the chair cushioned and the feet of the attendant slipped.

The food should be sufficient for the particular infant's needs, but if possible the bulk should be reduced.

The hours of feeding should be lengthened whenever practical. It is sometimes advisable to give a food that is partly predigested.

Extremes in the temperature at which the food is given must be avoided.

If beef blood or fruit juices have been a part of the diet they must be discontinued for the time.

At the time of feeding, and for fifteen minutes before and after, the room should be somewhat darkened while the baby lies in its crib.

In the cases that show a tendency not to yield promptly, it is advisable to begin the feeding with a medicine dropper or better still, with one of the feeders made especially for premature infants.

With such an apparatus, the rapidity with which the food is taken can be easily regulated and the effort of taking reduced.

The bromides and belladonna judiciously given are valuable adjuncts to the treatment, but the best results are obtained by an adequate adjustment of the food and the manner of its being taken and the complete subjection of the family and friends to the baby's interests.

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## PITFALLS IN DIAGNOSIS IN PEDIATRIC PRACTICE

By GODFREY R. PISEK

Professor of Diseases of Children in the New York Post-Graduate Medical School; Professor of Pediatrics in the University of Vermont College of Medicine  
New York

A large percentage of the general practitioner's practice embraces the care of children, and he is, as a rule, more adept in this branch (particularly if he has a liking for the little patients) than in any other specialty of medicine.

Pediatric practice demands that the physician be a close observer and then having gathered his information, that he should logically deduce his facts to form a conclusion.

The practitioner must approach his little patient divested of concepts which are applicable to adults; he must often be satisfied to make a diagnosis by a process of elimination, and to this end he must know what are the *probabilities* and *possibilities* in a given case.

It has been impressed upon the writer on many occasions that if these possibilities had been known, the diagnosis could have been made and a consultation perhaps rendered unnecessary.

This paper will attempt to classify the pitfalls which confront the practitioner and from which he can be saved if he is conversant with the possibilities and vagaries of the manifestations of disease in early life.

In *group 1* we may place those cases having *fever only* as the predominating symptom.

A newborn babe runs a temperature of 104 deg. F. or thereabouts, with no physical signs or discoverable symptoms. The cord is clean, the mother is normal and the baby has been put to the breast

and is apparently nursing. If the baby is weighed before and after such a nursing the fact will be disclosed that there is no increase in weight and that all the suckling efforts have been in vain; given one copious draught of breast milk from another suitable breast, and the temperature drops. We have been dealing with a case of inanition fever, a condition often mistaken for sepsis but which is readily corrected when appreciated.

A sudden high fever often without any referable pain or appearing in the course of what seemed a satisfactory convalescence in an infectious disease, should call to mind the possibility of an acute otitis media. We hear the statements—"there has been no discharge"—"there has been no earache"—"no pulling at the ears," therefore there has been no aural examination made. Routine examination of the drums is the only safe course to pursue, for a highly inflamed drum often confronts us in an unsuspected case. Early paracentesis furthermore saves many a mastoid involvement.

A serviceable auroscope should be part of the equipment of every man doing general practice. He need know none of the refinements of aural diagnosis; he should be able, however, to appreciate a normal from an acutely involved ear. The quick drop in temperature following puncture is extremely satisfactory to all concerned.

Perhaps the largest percentage of cases in this indefinite fever group will fall under the head of infection of the urinary tract. Fever with sharp rises and falls intermitting sufficiently to simulate malaria or atypical typhoid fever most generally occurs in female infants after the 7th or 8th month of life. A careful physical examination discloses no involvement of the lungs or endocardium, and no enlargement of the spleen. Unfortunately the high temperature soon disorders the digestive tract so that the stools become abnormal and the calomel and castor oil which are then usually given, still further complicate the picture. If pyelitis is thought of and a urinary sample is obtained, the diagnosis can be readily made from the milky, acid specimen containing numberless pus cells usually with myriads of colon bacilli.

In the face of a negative physical examination with irregular high temperature the practitioner is justified in securing a specimen by catheterization, if it cannot be obtained otherwise satisfactorily. Such a catheterized specimen taken under surgical precau-

tions and held in a sterile bottle will also be ready for the laboratory and from it an autogenous vaccine can be prepared and used in the event of failure of treatment with hexamethylenamine or the alkalies.

Malaria and typhoid fever in early life do not follow the classical lines laid down for them in text-books, and therefore may act as stumbling blocks. The intermittency in malaria is far from regular, the chills are not typical chills, and there may be no appreciable period of sweating.

In typhoid, we may see no characteristic pea-soup stools; on the contrary constipation may be present. Enlargement of the spleen may not occur or only very late in the disease, and the roseola spots may never be seen. In both conditions the blood examination must be depended upon for a definite diagnosis. In fact the diagnosis of malaria without the presence of protozoal blood infection is untenable.

Influenza and an infected adenoid mass are also to be reckoned with as possibilities giving few signs other than the irregular febrile course; but in the main, if we recollect, the U—B—E. U for urine—(pyelitis) B for blood (in malaria and typhoid) and E for ears, we will uncover the greater number of these seemingly obscure febrile cases.

In *group 2*, we may place those conditions which *do* give evidence or symptoms besides fever, but the symptoms of which are misleading. A retro-pharyngeal abscess, for example, has been mistaken for laryngeal diphtheria, pneumonia and meningitis. The muffled blubbery voice or cry is quite characteristic if one studies well the different cries of child life; although the dysphagia, the retracted head and respiratory difficulty quickly induced when the head is bent upon the chest should lead us to think of the possibility of a retro-pharyngeal abscess, and examine the throat under the best possible conditions of light. A bulging centralized mass, giving evidences of fluctuation to the palpating finger is sufficient warranty for incision and drainage.

Empyema is more rarely missed than formerly; but still the teaching based on adult chests, namely, the demand for absolute flatness, loss of respiratory murmur, Skoda's sign, etc., lead to delay if not failure of diagnosis.

We are warranted in making exploratory puncture in a delayed pneumonic convalescence, when we obtain over a localized area, dulness to flatness, diminished or distant breath sounds, and resistance to the percussing finger. Nor should we limit ourselves to the so-called classical area for puncture; aspirate over the site of the abscess in any region not precluded by an underlying organ other than the lungs. A good sized needle with a powerful syringe behind it must be used, otherwise fibrino-plastic exudate may interfere with the finding of pus.

The quite general employment of pasteurized milk without the prophylactic use of orange juice, and the undiminished consumption of condensed milk and the proprietary foods bring their crop of scorbutus cases which are most insidious particularly in their inception.

The child may be willing to play lying on its back and using its hands, but cries immediately and violently when handled; diapering particularly is painful. The tibial tenderness and the spongy bluish gums seen only perhaps around the two erupted teeth should be sufficient for a therapeutic test. The history is more often misleading than helpful, unless the condition is specifically in mind and the questions are directed along this line.

Severe unrecognized cases are apt to be mistaken for osteomyelitis, or epiphyseal fracture and often are directed to the surgeon's hands.

A rarer condition fortunately, but which we meet with in epidemic waves during some winters is glandular fever, undoubtedly an infection emanating from the naso-pharynx and involving the neighboring lymph glands unilaterally at first, later symmetrically. A rise from 103 to 105 deg. F. precedes each involvement of a new glandular chain. The diagnosis once made it is satisfactory to know that the course is self limited and that except for an occasional nephritis there are no complications to fear.

*Group 3.* This group of cases give *evidence of their presence* but these symptoms are *obscured by others* found in commoner conditions.

The baby with a congenital pyloric obstruction, slowly but steadily loses weight, vomits frequently and regularly and is constipated. Before the true diagnosis is made, in the great majority of cases,

the baby is taken off the breast, if previously breast fed, because it is doing badly on its mother's milk; it is then run through various milk modifications, but still persists in vomiting, the proprietaries are then resorted to in spite of the doctor's better judgment, and still the baby vomits and emaciates to a degree. Vomiting directly after ingestion of the pabulum; small, very small stools, and loss of weight especially in a breast fed infant should lead us to examine for a peristaltic wave which will pass from left to right across the greater curvature and to palpate for a pyloric tumor.

The X-ray can be depended upon to differentiate a case of congenital tumor from one of pyloro-spasm as the writer has demonstrated in a previous article,\* and thus separate those cases which are suitable for medical treatment from those which should be placed at the earliest possible moment in the hands of the surgeon. The Ramstadt operation is a great step in advance and can be performed with little danger and is most satisfactory in its results.

Intussusception in early life reaps a high mortality because the cases are not brought to the surgeon's hands within the period of safety, that is, the 48 hour limit. The diagnosis is delayed because of misleading symptoms and because the physician oftentimes does not himself examine the stools, taking the attendant's report on the dejecta. These are described as containing fecal matter while in fact they are composed only of mucus and blood. The periods of calm which ensue after the attacks of pain and shock are also very apt to throw the physician off his guard. Castor oil has probably been given, and at the next visit the baby is fairly happy and even may be induced to smile or play. Suspicion of obstruction may then be discarded until the doctor is hurriedly recalled by the advent of another attack of pain and perhaps collapse. Early diagnosis and the abandonment of mechanical efforts to reduce the intussusception will change materially this unnecessarily high death rate. The finding of a tumor should not be the main consideration; one must be impressed with the fact that a well child has suddenly been plunged into a condition of pain and shock; that the stools have materially changed in character and that vomiting persists in spite of abstinence from food.

\*Pyloric obstruction, with a comparative study of the normal stomach of infants. Arch. Ped., Vol. xxix, No. 12.



Another condition which can now be very satisfactorily treated also falls into this group. It occurs in the newborn and is generally unrecognized because nurses consider it their duty to hurry away or hide the soiled diapers at the advent of the doctor's visit. Dark grumous blood admixed with the meconium may easily pass unrecognized by the inexperienced attendant. *Melena neonatorum* is the commonest form of the spontaneous hemorrhages of the newborn, and we are dependent upon this evidence from the intestinal tract for its detection.

The bleeding is readily stopped by the injection of human blood serum or whole blood from a homologous donor, and by its use a life can be saved. The use of coagulin or horse serum are not advised except as temporary expedients, until the human serum or blood can be obtained; otherwise disappointment may occur and valuable time will be lost.

The last group deals with developmental defects, associated with some degree of mental retardation.

The writer's experience leads him to the belief that cases of infantilism, cretinism, achondroplasia and mongolism are not closely enough differentiated and that they are all apt to be considered as amenable to thyroid medication. As the cretins alone respond to the use of thyroid, we see disappointed parents seeking aid elsewhere. Mongolism more closely simulates types of cretinism but can be distinguished, if one notes the lack of the protruded tongue, the lordosis and stumpy extremities of the cretin, but finds wide apart slightly slanted eyes, a brachycephalic head with asymmetrical ears, a narrow high arched palate, irregular dentition and characteristic trident hands. These cases respond to somewhat higher mental tests than the cretins, but have no idea of the abstract.

The achondroplasiacs or dwarfs can be readily distinguished by their disproportionate body and extremities. These latter are short and often show rachitic changes. If the humeri and femora are both disproportionately short, and the head is large resembling that of a hydrocephaloid, the diagnosis is established.

The glandular extracts, particularly those having a bearing on the processes of development, such as the pineal structure, may be given with the hope of influencing this condition and suitable cases of infantilism.

We are still using the endocrine therapy with the exception of the thyroid extract rather empirically but further study and experimentation by the profession offer hope of reward.

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## THE THERAPEUTIC DIAGNOSIS OF AMEBIC ABSCESS OF THE LIVER

By WILLIAM ALLAN

Charlotte, N. C.

Liver abscess, particularly the solitary abscess, is now admittedly due to amebic infection in nearly all instances. The wide prevalence of amebic dysentery over the southern half of the United States is becoming pretty generally recognized, and the fact that amebic infection is by no means rare in the northern half of this country has recently been pointed out by several observers. So that in any consideration of the diagnosis of liver abscess it is important as a preliminary step to insist that this lesion, at least in the South, is an every-day affair and not a disease of foreign parts. Undoubtedly the greatest obstacle to the diagnosis of amebic abscess at the present time is this lack of appreciation that it is one of our common maladies. A good idea of the prevalence of abscess may be gained from Elliott's (1) report of 116 cases from the New Orleans hospitals within the short period of five years.

The full-blown abscess with pain over an enlarged tender liver, with septic fever, chills, sweats, and leukocytosis, possibly with dry, hacking cough or slight jaundice, associated with loss of flesh and dysentery, or a history of recent dysentery, does not present a difficult problem. However, the clinical picture is rarely complete and the patient may complain only of chills and fever, with or without pain. Enlargement of the liver cannot always be determined.

Fever is one of the earliest and most constant signs of hepatic abscess, and is very generally accompanied by leukocytosis with relative increase in the polymorphonuclear neutrophils. Any type of fever may be present, and in large abscesses the temperature may be normal. Pain, though not so early a symptom as fever, is

fairly constant, occurring 16 out of 19 times with me. The pain may be a steady soreness or become intermittently sharp when the parietal peritoneum becomes involved. Involvement of the diaphragm produces a hacking, dry cough, and occasionally distressing singultus. The pain of liver abscess is usually referred to the right upper abdominal quadrant extending up over the lower ribs, or to the right axilla. Occasionally, as in gall-stones, the pain may be referred to the right scapula or to the top of the shoulder.

Increase in the size of the liver, either up or down, is the most significant of the physical findings. There is very generally tenderness to pressure and percussion over the enlarged liver, with discomfort when lying on the left side, as in right pleural effusion. Occasionally there is slight jaundice. When bloody dysentery is present, or has recently been present, the chances of liver abscess are very much enhanced, though in a considerable number of cases there has been no dysentery and in many no amebæ can be found in the stools.

We are usually presented clinically with the problem of finding the cause for fever and pain in the right side, possibly with chills and cough. The physical examination may reveal the signs of infiltration at the base of the right lung. Considerable enlargement of the liver upward may simulate pleural effusion. A needle will eliminate the latter, or better still, when possible, Röntgen examination will show the right leaf of the diaphragm elevated and its respiratory excursion considerably reduced. If enlargement of the liver is found on physical examination the probability of abscess is strong, for in comparison with abscess the other causes of enlarged liver (aside from evident cardiac decompensation) are infrequent. As in any other country where malaria is a possibility, examination of the blood is as indispensable as physical examination in every patient with temperature above normal. Liver abscess will almost invariably show leukocytosis or increased polynuclears, usually both. Urinalysis is without any special significance. Examination of the feces is fortunately gradually becoming a routine measure in southern hospitals. If amebæ are found in the stools it becomes necessary to differentiate these organisms as a large part of the population in many localities carries the harmless *endameba coli*.

Finding *endameba histolytica* depends not only on the presence of amebic colitis, but also on the experience of the observer, preliminary purgation in the absence of dysentery, the state of activity of the ulcerative process, the physical and chemical conditions of the stool, etc.

After our facts have been assembled in any case it, of course, becomes necessary to eliminate the possibilities other than liver abscess. The blood examination eliminates at once malaria and tuberculosis unless these diseases are present as complications. In more than 80 per cent. of the cases pain directs attention to the region of the liver. With upward enlargement of the liver the needle or Röntgen ray will clear the pleura of suspicion. With enlargement of the liver downward we must consider pyogenic liver abscess from appendix or gall-bladder, the usual causes of pus in the right upper quadrant, and enlargement of the liver from syphilis, neoplasm, and cirrhosis. Pyogenic infections either in or out of the liver can be differentiated from amebic abscess by the administration of emetine, as a large per cent. of the latter subside completely under the influence of this drug (2).

That livers enlarged from other causes may also harbor amebic infection is shown by Achard and Fox's report (3) of two cases of hypertrophic cirrhosis in which multiple amebic abscesses were also present. In 1910 I saw a negro man from New Orleans who came into the dispensary complaining of a soreness in the abdomen and aching shins. He had irregular attacks of flux, fever, and chills and presented an enormous tender, bulging liver. He refused both operation and aspiration, and after one dose of ipecac pills refused further treatment. After reporting occasionally for eighteen months he wandered off, but returned in 1916 with a history of having coughed up his abscess a few months previously, and instead of the bulging, painful liver reaching below the umbilicus, he presented a firm, painless liver reaching half way to the umbilicus. A positive Wassermann and increased leg pains made it seem very likely that the remaining hepatic enlargement was due to syphilis.

To clear up a doubtful diagnosis the aspirating needle or exploratory operation are usually recommended, search being made in the pus for amebæ. Even when the diagnosis is not in doubt, it has

been and still is (4) customary to evacuate and drain these abscesses and to give emetine after operation if amebæ are found. At the present time it seems to me that the use of the aspirating needle and the knife, whether for diagnostic or therapeutic purposes, is unjustifiable until medicinal measures have been given a careful trial. Emetine will settle a doubtful diagnosis more often than exploratory puncture, and will cure more amebic abscesses than surgery.

The partial response of amebic abscess to quinine is often very striking, and in the absence of blood examination frequently leads to diagnosis of malaria. In February, 1911, I saw a middle-aged white man who had been complaining of fever and dumb chills with some pain under his right scapula off and on for five months. During this time he had been given several courses of quinine, and each time his symptoms had subsided entirely for a week or so. He had had no dysentery and his liver was only one finger below the costal margin. Later a small abscess high in the right lobe near the posterior surface was opened and drained. In November, 1916, one of my friends told me his nurse had been having chills all summer and in the mountains of this State had been treated for both typhoid and malaria. Quinine in large doses for ten days would always break up the chills, but as soon as the girl stopped the quinine the chills returned. This history of partial response to quinine suggested amebic abscess, and although neither enlarged liver nor dysentery was present, emetine stopped the chills promptly and permanently, as recited below.

Emetine is a particularly valuable diagnostic agent in differentiating those cases of liver abscess without enlargement of the liver and without dysentery, as illustrated in the following case histories. We are called upon to find the cause for an irregular fever with leukocytosis; pain and cough may or may not be present.

CASE I. A white woman, age 50, housewife, was seen December 3, 1916, with fever and jaundice. She had had a continued fever for a month and jaundice for a week. There were no other symptoms, and physical examination revealed nothing to account for her fever. There was no pain, no cough, no dysentery, and no enlargement of the liver. Urine negative. White count 24,000 with 90 per cent. polynuclears. Stools negative. Emetine daily

for a week without benefit. Prompt recovery after draining the gall-bladder.

CASE 1. A white woman, age 50, housewife, was seen December 1916, complaining of chills. She had been having chills about every second two weeks since April and had been taking quinine pretty steadily. There was a dry, hacking cough, but no complaint of pain. Physical examination entirely negative except for tenderness around right costal margin. Liver not palpable. Urine and feces negative. White count 16,000 with 80 per cent. polynuclears. Is having chills daily. Given from one to two grains of emetine daily and since the first dose has had no more chills or cough (eight months ago).

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## THE TURNING TEST IN APPENDICITIS

By SILAS C. BLAISDELL

Brooklyn-New York

I believe this test to be original with me, having employed it for the past twelve or fourteen years. If it is not original it is a tale that bears telling twice.

When you enter a room where there is a case of suspected appendicular trouble you will rarely, if ever, be the attack acute or otherwise, find the patient lying on the left side. He will almost always be lying on his back or on the right side. You ask him why he lies on his back or right side and he replies that it hurts him when he turns on the left side. That was the first thing which made me ask "Why is that?" Since that time it has always been my procedure to use this turning test and in ninety per cent. of all appendicular troubles, or those involving an inflammatory condition of the appendix, *the pain is increased when the patient is turned to the left and diminished when he is turned back on his right side.* The pain is not so intense when the patient is on his back

as on the left side. If every other symptom is absent I always advise operation if that test is marked and in over ninety per cent. of my cases it has proved to be correct.

In making the test the abdomen must not be allowed to touch the bed thereby allowing the viscera no support when it pulls to the left and drags along the sensitive appendix thereby causing additional pain. With a rapid pulse, with or without temperature, and the turning test present, operate.

I start my incision technically two fingers' breadth away from the umbilicus and one finger's breadth below the level of the same running from the right downward and to the left, cutting through the skin, superficial and deep fascia and also through the anterior sheath of the rectus in the same direction. The knife is turned, the handle splits through the rectus (the rectus is not cut), the transversalis fascia is picked up quickly and a nick about one inch in length made therein, and a little nick is also made in the peritoneum just large enough to insert one finger. With a stick sponge I push the omentum out of the way, insert two fingers into the opening and lift up the cecum. After removing the appendix I close the wound with a circular stitch which takes in all the layers.

I believe that every row of sutures we insert is a nucleus for infection and causes pus and we should endeavor to make these as few as possible.

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## ABDOMINAL PAIN: ITS DIFFERENTIAL DIAGNOSIS

By ROBERT FRANKLIN IVES

Brooklyn-New York

The successful comprehension of abdominal ailments, with their manifold methods of expression, demands an intimate acquaintance with the anatomy, physiology, histology and pathology of the abdominal structures and organs. In fact, success in the diagnosis of this complexity can only be obtained by observing the greatest care in the anamnesis, and after the most careful and painstaking examination, and with full laboratory data.

Nothing must be taken for granted; and all examinations must

be made with the fullest exposure of the abdominal area, and a careful general survey of the body as a whole.

Preconceived ideas must not bias judgment, or the importunities of the patient or friends for relief, especially in the acute pains, distract from the vital necessity of knowing the cause of the pain. Wait! Take ample time, and never hurry unduly is an abdominal axiom of great importance.

Especially, do not administer an opiate until you are sure that you are not clouding your symptoms, and so preventing a diagnosis.

Express the diagnosis only after the same has formulated itself, and that after the most exacting differentiation.

The recent progress in diagnostic methods, coupled with the knowledge that the autopsy statistics gathered by Cabot at the Massachusetts General Hospital show a remarkable tendency to error in abdominal diagnosis, and the tendency to apply the easier method of making the diagnosis after exploratory or operative celiotomy in many obscure cases is sufficient reason for the presentation of this paper.

Better is it to adopt the method used by the Mayos at Rochester, or by the late John Murphy, of Chicago, and all noted clinicians—whose methods embrace a complete physical examination in addition to the local—where each case is given exact individual study in order to prevent error in judgment.

The hypothesis developed by Dr. James Mackenzie, of London, while not perfect, sheds much light upon the interpretation of abdominal symptoms. I refer to the so-called viscerosensory, and the visceromotor reflexes. They include such sensory phenomena as pain, hyperalgesia, and rigidity of the tissues, muscles and skin. The accurate noting of these less obtrusive phenomena will often throw a flood of light on many an obscure process, and reveal the mechanism by which they work and the site of the lesion. They are, as stated, not absolute, but very suggestive.

The board-like rigidity of the rectus, and upper abdominal muscles following the perforation of a duodenal ulcer; and the rigidity of the muscular wall over the site of an inflamed appendix are examples of these reflexes.

Pain in distant areas, or the so-called referred pains, must always be borne in mind: the true association of which may prove *very*



helpful. The reason why these pains are referred to portions of the body so distant is because, in the course of development, the tissues that in a low scale of life immediately covered the organ have been displaced. Thus the pain felt in the testicle in renal colic is due to the fact that in the journey down to the scrotum, the coverings of the testicle receive a twig from the first lumbar nerve—thus, when the center of this nerve is irritated or stimulated, as in renal colic, the pain radiates to the testicle, a pathognomonic sign of renal trouble.

Likewise, in the condition of coronary sclerosis (angina pectoris) the referred pain often passes to the epigastrium, instead of into the axilla or down the inside of the arm. This must always be kept in mind, or the true diagnosis of the myocardial exhaustion may be overlooked.

In gall-stone disease shoulder pain is not an infrequent complaint, even extending down the arm. It may persist with such severity that the causal condition may be overlooked, and the case considered as one of neuritis—the expulsion of a gall-stone being followed by instant relief.

Individual idiosyncrasy modifies the subjective symptoms of pain; thus it is well to remember that some nervous people may call a normal physiological peristaltic wave *pain*, while the more plethoric or stoical may minimize it as an important symptom.

I would advise the development of a technic in eliciting the history and the localization of the pain. The schemes elaborated by Lockwood, Bassler and others show the importance of systemized data. Lockwood's scheme of locating pain is of value, and should be used in the chronic types.

This analysis of pain should be made, *in order to associate it with some organ*:

Is it constant or intermittent (periodic)?

If constant, does it have seasonable variations? (Remember about 95 per cent. are periodic.)

Is it local or diffuse?

If local, does it radiate?

Is it acute or chronic?

Does the pain relate to any physiological act, as eating or standing?

Is it present on awakening?

Does it occur after meals?

Is it sharp, cutting or diffuse?

Is it gnawing or burning?

Is it relieved by food or soda?

Has it associated symptoms? Nausea—vomiting—headache—chest pain—fever—sweats—chills—dyspnea—pallor—cyanosis—icterus?

Has it produced shock?

Especially would I call attention to a definite group of symptoms (syndrome)—the “pyloric syndrome.” It is of the greatest importance, and its significance illuminates the subject greatly. Herz, of London, proved that tension causes pain. The presence of food or liquid in the stomach creates tension, produces secretion, stimulates peristalsis, and increases the hypertonic muscular contractions, with the production in some conditions of a pylorospasm. When, therefore, diseased reflexes storm the solar plexus, these elements become pronounced and productive of painful sensations and are called *pain*.

The importance of this syndrome lies in the fact that it is excited into being not only by the presence of an ulcer of the stomach or duodenum, but by disturbances in the appendix, gall-bladder, kidney or spleen. It may be produced by the presence of adhesions in some portion of the intestinal tract, either congenital or inflammatory, as a result of a neurosis, or as a result of Bright’s disease or tuberculosis. It thus explains many cases diagnosed as ulcer or operated upon without finding ulcer present. To recall this truth will prevent the making of a snap diagnosis of ulceration, and will awaken the faculties to precision.

With this introduction, let me review some of the diseases in which abdominal pain plays its part. The list is instructive in opening our eyes to the need of careful differentiation. They are as follows:

Skin:

Painful in erysipelas.

Herpes zoster abdominalis.

Peritoneum:

Acute or chronic peritonitis.

Tuberculous peritonitis.

**Pulmonary:**

Reflected pain in pleurisy or pneumonia.  
Especially present in children.

**Digestive:**

Cardiospasm (spasm of the esophagus).  
Gastralgia (gastric spasm).  
Gastric ulcer.  
Acute, chronic, perforating, and with complications.  
Impure or unripe food gastritis.  
Duodenitis.  
Gastric carcinoma.  
Colitis.  
Appendicitis.  
Gastric erythrim.  
Functional gastric disturbances.  
Hyperchlorhydria or neurotic motor irritability.  
Hour-glass stomach.  
Pyloric stenosis.  
Duodenal ulcer.  
Acute, chronic, perforating, and with complications.

**General:**

Cecal stasis.  
Enteroptosis.  
Gastroptosis.  
Nephroptosis.  
Acute dilatation (gastrextasis).  
Plumbism.

**Liver:**

Cirrhosis.  
Alcoholic.  
Luetic.  
Secondary biliary cirrhosis.  
Hanot's hypertrophic cirrhosis.  
Pick's pericarditic pseudocirrhosis.  
Hepatitis.  
Acute yellow atrophy.  
Abscess—cyst—gummata.  
Subphrenic abscess.  
Intrahepatic primary carcinoma.  
Intrahepatic secondary carcinoma.  
Inflammatory processes in the capsule.  
Cholangitis.

**Liver vessels:**

Aneurism of hepatic artery.  
Thrombosis of portal vein.

## Gall-bladder :

Cholecystitis.

Cholelithiasis.

Stone in fundus of gall-bladder.

Stone in neck of gall-bladder.

Stone in cystic duct.

Stone in cystic duct at the junction with the hepatic duct.

Stone in the duodenal portion of the common duct.

Stone in the papilla of Vater.

Stone in the common hepatic duct.

Carcinoma of the papilla of Vater.

Ascaris in common duct.

## Pancreas :

Pancreatic cyst.

Carcinoma of head of pancreas.

Acute suppurative pancreatitis.

Acute hemorrhagic pancreatitis.

Pancreatic calculi.

## Kidney :

Infarct.

Embolism renal artery.

Dietl's crisis (movable kidney).

Ureteral spasm from aberrant artery.

Nephritis.

Pyelitis.

Pyelonephritis.

Pyonephrosis.

Hydronephrosis.

Perinephritis.

Renal congestion.

Renal tuberculosis.

Paroxysmal hemoglobinuria.

New growths.

Hypernephroma.

Sarcoma.

Carcinoma.

Tuberculoma.

Renal calculi.

Body—pelvis—ureters.

Blood clots (renal)

## Spleen :

Toxic or infectious.

Malaria or syphilis.

Perisplenitis.

Infarcts.

- Splenomegalia.
  - Gaucher type.
  - Von Jaksch's pseudoleukemic.
- Anemia of infants.
  - Hemolytic jaundice.
  - Acquired: Hayem-Widal type.
  - Congenital: Chauffard-Minkowski type.
- Banti's disease.
- Pernicious anemia.
- Intestinal obstruction and inflammations:
  - Invaginations.
  - Bands-kinks—adhesions—membranes.
  - Volvulus.
  - By calculi (obstipational ileus).
  - Constipational colics.
  - Ileocolitis.
  - New growths.
    - Carcinoma.
    - Sarcoma.
    - Tuberculoma.
- Blood vessels:
  - Angina abdominis.
  - Aneurisms abdominal aorta.
  - Thrombosis.
    - Inferior vena cava.
  - Embolism.
    - Superior and inferior mesenteric.
- Abdominal hernias:
  - Umbilical.
- Strangulatory:
  - Inguinal.
  - Femoral.
  - Retroperitoneal.
  - Mesenteric.
  - Diaphragmatic.
- Pelvis:
  - Pregnancy—labor.
  - Pregnancy—abortion.
  - Pregnancy—ectopic.
  - Ovaritis.
  - Salpingitis.
  - Salpingo-oophoritis.
  - Pelvic peritonitis.
  - Pelvic abscess.
  - Pelvic adhesions.

Fibroids.  
Ovarian cysts.  
Dermoid cysts.

Etc.

Such a list is undoubtedly impressive and yet does not completely include all medical and none of the traumatic surgical causes of pain.

As it is manifestly impossible to review all these causes differentially, it may be wise to analyze a few of them, somewhat in groups:

#### THE ULCER GROUP.

The ulcer group seldom causes great difficulty of detection, provided we ever keep in mind the significance of the pyloric syndrome and do not diagnose ulcer when none is present.

The pain of gastric ulcer usually begins shortly after the entrance of food in the stomach, and gradually increases until it reaches a climax. It is often associated with, and for a time relieved by, vomiting. Its character is dull, boring and is located at the epigastrium. It penetrates through to the back, and develops at times a tender spot at the tenth, eleventh or twelfth space to the left of the spine. Gastric ulcer cases are apt to cause great tenderness over the epigastric region. These patients refuse to tolerate even the slightest pressure locally. The vomitus may be bloody or contain blood. Anemia is common, while pyrosis or regurgitation of a sour-tasting fluid frequently occurs. In fact, belching and acid regurgitation with the other signs are pathognomonic of *ulcer ventriculi*.

The differential from duodenal ulcer is rarely difficult. The striking and characteristic symptomatology of the latter, with its late pain, relieved by food and soda, with melena instead of hematemesis, is important. Orthoform, grs. xv, will promptly relieve gastric ulceration pain and not affect the duodenal type.

#### PERFORATION.

The pain of perforation of a gastric or duodenal ulceration is startling in its severity, and calls for accuracy in the differential. When the eroding process penetrates the walls of the stomach or gut without a limiting perigastritis—or periduodenitis—there oc-

curs one of the most dramatic and fatal complications of ulcer—perforation with resulting peritonitis. The *intense* pain in the upper abdomen, agonizing in severity, overshadowing every other symptom—there is great depression, *but no real shock*. After a time the pain becomes less atrocious, and its lessening severity may thus deceive. The board-like rigidity of the abdominal wall, however, tells the story. A febrile condition supervenes, and an increased leukocyte count helps to make the diagnosis. Operation following the primary depression is imperative.

The differential count must always exclude appendix and gall-bladder, which can readily be done by means of a careful history of preceding symptoms.

The symptoms of perforation of gastric and duodenal ulcer are similar. The preceding history must make the differential diagnosis between them.

*Acute hemorrhagic pancreatitis* stands as a bugbear in the minds of many physicians, but its differentiation is both easy and accurate.

This and a ruptured pancreatic cyst are sudden in onset and *intense* in character, and resemble perforating ulcer. The pain, however, is more diffuse; the area of resistance and tenderness extends *transversely* across the abdomen just above the navel, and describes the region of the pancreas. The signs of peritonitis *fail to appear*. *The shock is greater; the prostration more marked*. The pulse at once shows a poor quality. Vomiting is frequent; *cyanosis* appears, and death early closes the scene. Hemorrhagic pancreatitis attacks corpulent people or women in the early months of pregnancy. It rarely accurs before middle life.

#### GALL-BLADDER DISEASE.

The pain of gall-bladder disease is due to overdistension of the walls, to excessive contraction of its muscular walls, or to irritation of the mural nerves.

Overdistension may occur from obstruction of the cystic or common ducts by inflammation, stone, pancreatic lesions, pressure from adjacent lesions in viscera, and tumors. The organ enlarges, becomes very painful, and is readily felt by palpation. If *cholecystitis* or infection occurs, there is fever, chills and great tenderness. The diagnosis is assisted by Murphy's gall-bladder percussion

method, by palpation of the enlarged organ, and by the urobilinogen test found in the urine. Remember that chills and fever signify an infection.

*Perforation* should always be considered and prevented by operation. The Murphy technic is as follows: Having previously removed all clothing from the part to be examined, place the patient in a sitting position, bent well forward with hands resting on the knees. The examiner stands behind and places his hand with palm flatly against the abdomen, immediately below the rib on the right side. Have the patient breathe deeply. After each expiration the examiner's hand follows the abdominal wall until it approaches the gall-bladder, when suddenly the respiration ceases, and with a gasp or grunt the patient complains of severe pain.

Murphy also elaborated a finger percussion sign of value, viz.: While the patient is lying, place the left hand over the area of the ninth rib, the fingers lying over the gall-bladder. Have the patient breathe deeply and forcibly percuss the bent and stiffened fingers. If inflamed, *an agonizing pain results*.

The sharp, cutting pain of *gall-stones* radiating to shoulder and down into the abdomen, with vomiting, and no fever—located over the gall-bladder—with or without icterus—and the negative ulcer signs, readily differentiates cholelithiasis. Gall-stone pain is apt to occur at night or some hours after eating. The sudden onset and cessation with local tenderness over the gall-bladder are evident signs.

#### CHARCOT'S INTERMITTENT FEVER.

Charcot's intermittent fever as a result of impaction in the cystic duct should always be kept in mind. This Charcot's fever is characteristic. The patient may have pain suggesting cholelithiasis with a sudden rise of temperature to 103 deg. F.-104 deg. F., to fall to normal within a few hours.

#### DIETL'S CRISIS.

The differentiation from Dietl's crisis or renal colic may at times confuse, but the reflected pain of renal colic to the testicle and the localization to the lumbar region soon tells the truth. *Dietl's crisis* does not produce the pain over the gall-bladder, and as a rule the tender kidney can be felt by palpation.



## CHARCOT'S GASTRIC CRISIS.

I would like to call attention to that confusing entity, *Charcot's gastric crisis*. These attacks, when typical, are characterized by a *sudden, intense abdominal pain*, affecting the epigastrium, or lower part of the sternum, and radiating to the back and shoulders, usually accompanied by pain in the lower extremities, or by a distinct girdle sensation. The gastric unrest terminates in vomiting of a highly *acid* fluid; the vomiting is difficult and accompanied by retching. Temporary relief follows the vomiting. Excessive salivation and sweating accompany the above. The pain is unbearable. The patient tosses about in distress. The differentiation is made by finding the evidence of *tabes dorsalis*. This picture should always be remembered, and *every* patient tested by the Wassermann reaction.

## PLUMBISM.

In these days when the automobile flourishes, the occupational disease termed plumbism or lead colic must ever be prominent in the diagnostic mind of the physician. It is a diagnosis of great importance, and hinges on our knowledge of lead poisoning.

The patient complains of *intense, knife-like pain* centering around the navel, radiating in all directions, with vomiting and constipation. There is abdominal tenderness, but *no fever*. The colic may last for hours, or for weeks. The abdomen is hard and contracted. The patient usually is seen bending over, pressing hard against the abdomen, which gives some relief. Anemia, pain, contracted belly, constipation, high pressure pulse and *no fever*—especially if showing the Burton's line on the gums—should lead to the suspicion of lead poisoning.

A blood examination reveals the basophilic granulation. This consists in the development in the erythrocytes of granules which stain with certain basic stains. The best stain to use is thionin stain, which shows an intense blue.

The lead line is found in only 50 per cent. of the cases, but when present is pathognomonic.

## THORACIC DISEASES.

Abdominal pain frequently simulating appendicitis occurs in thoracic diseases. Therefore, in every question involving the ap-

pendix, attention should be directed to the thorax, especially in *children*.

#### CECAL STASIS.

I wish to direct attention to a condition which in my judgment is of paramount importance, which is seldom diagnosed, is very common, and which is the cause of much illness. I refer to *cecal stasis*.

The alimentary canal possesses two pouches or reservoirs: the stomach, which receives, and for a while retains, the food at the beginning of digestion, and the cecum, which receives, and for a while retains, the remnants of digestion; when for the most part digestion is complete. In either of these there may occur motor insufficiency, either from atony or overtonicity. The cecum normally retains the contents in order that imperfectly digested particles may undergo further separation, hydration and absorption. As a result, the intestinal cecal contents gain in consistency, thus offering mechanical stimulation to the cecum, and the expulsive peristaltic wave moves the mass upward into the ascending colon. Bacteria abound and putrefaction is common.

As a result of stasis, there occurs in patients periodical attacks of *severe pain* with fever, tenderness over the appendix, anorexia, nausea, vomiting and coated tongue. In a word, symptoms of *auto-intoxication*. The condition is repeatedly diagnosed as appendicitis, and after the appendectomy, to the chagrin of the doctor and surgeon, the condition persists in all its severity. Difficulty may be experienced in the acute stage, but the differential may be made by the *absence* of an *increased leukocytosis*, which is present in appendicitis.

May I insist here that every physician should be prepared to make, at any time, his own blood-corpuscle and differential count? Only by so doing can a diagnosis be made without undue delay.

It is in the cecal cases that Arbuthnot Lane's recommendations for Russian oil find a helpful field of response.

#### APPENDICITIS.

Shall I take time to mention the surgeon's friend, the appendix? I will simply quote the 10 per cent. mortality rate to-day in most

of the best hospitals to emphasize the need of prompt diagnosis and as prompt treatment by operation.

A case which starts with colicky pain, nausea, vomiting, elevation of temperature, may develop a leukocytosis and local sensitiveness in the right flank in six or eight hours. In 24 hours the pain and fever may be gone, because a gangrenous appendix causes no pain—its nerves are dead. It produces no elevation of temperature or leukocytosis because absorption of the products of infection are impossible through a dead mucosa, with the resultant drop in the previous temperature. To quote the late J. B. Murphy, "The sudden disappearance of pain is the last call to operate with safety; the next symptom will be rupture and peritonitis, and perhaps a fatal ending."

Always search for tenderness and don't be deceived.

I would call attention to a point mentioned by Robert T. Morris that lies on the same line on which is found McBurney's point, that proves highly helpful in differentiation. On a line drawn from the anterior superior spine of the ilium to the navel is found McBurney's point, one and one-half inches from the anterior superior spine. Another point, one and one-half inches from the navel on the same line, lies over the right lumbar ganglion of the sympathetic nervous system, and tenderness of these ganglia prove of great diagnostic value.

The thought to be impressed is this: In acute attacks of the appendix McBurney's point is most tender, with tenderness also over the right lumbar (Morris') point, and *no tenderness* usually over the left lumbar (Morris') point.

In post-inflammatory processes in and about the appendix, when scar tissues or adhesions have formed the chronic appendicitis cases, the tenderness over McBurney's point may disappear, to increase over Morris point with *no tenderness on the left side*.

Again: When the appendix is kept congested without infection, as is found in cases of loose right kidney, there is no tenderness at McBurney's point, but persistent tenderness at the Morris point mentioned. This is worth remembering.

When the right Fallopian tube is involved as a result of original appendiceal inflammation, the Morris' point proves very tender with no tenderness on the left side. *Again: When the Fallopian tubes*

*are inflamed, and not the appendix, then both right and left Morris' points are tender, but not McBurney's point.*

To recapitulate:

The patient comes to us with the appendix a question mark.

If right lumbar ganglion (Morris' point) is tender alone—then appendix trouble. Right and left lumbar ganglia together—pelvic trouble. Trouble is cephalad when neither right nor left is tender. These points have been of very great assistance to me in cases of obscure diagnosis.

#### INTESTINAL OBSTRUCTION.

A sudden abdominal pain, associated with vomiting, especially if repeated, should always suggest an acute abdominal lesion, such as obstruction by volvulus, bands, intussusception, strangulated hernia or obstruction by a foreign body—obturatorial ileus, etc. If the vomiting persists and the pain gradually increases and spreads over the abdomen, it is certain that the lesion is one of increasing gravity. These cases must positively be accurately diagnosed and promptly treated, and *should never be relieved by opiates to cover the diagnosis*, hoping at the next visitation to be able to fathom the etiology.

Should a chill with rising temperature appear, it is very likely that the trouble is of an inflammatory nature.

If instead, the pain is paroxysmal without temperature, colic or obstruction should be sought.

If the colic is gripping and with loose stools without blood at first, enteritis is probable.

Should slimy or mucus stools with blood, and the case an infant, then intussusception should be considered.

In acute pain, without tumor or mucus or bloody stools, with an area of exquisite tenderness or rigidity, that of perforation, as in typhoid or ulceration, is sought.

Volvulus *always* develops a palpable tumor.

Remember that borborygmus and active visible peristalsis are important symptoms of *chronic intestinal obstruction*, such as malignant neoplasm of the colon.

Remember that new growths of the sigmoid can usually be felt in the left iliac region, while new growth of the rectum can be felt with the examining finger, or seen with the proctoscope. Therefore, always examine and re-examine patients.

It is well to recall that cancer of these areas does not show blood in the stools until ulceration results—hence, most reliance must be placed upon the borborygmi and active peristalsis.

Remember the bowel is silent in paralytic ileus, and noisy in obstructive ileus, and remember that mechanical obstruction does not have initial elevation of temperature.

Obstruction caused by bands always suggests a previous operation or an inflammatory history, and remember from a prognostic import that when an obstruction is of several days' duration, the great question to settle is whether it is a strangulation or an obturation ileus.

If a patient with strangulation from volvulus, band or hernia—they become cold and clammy, and exhibit the signs of shock, and are nearly moribund in four days. On the contrary, if the patient's hands and feet are warm, without marked shock, the trouble is rather mechanical or obturational than strangulatory. This fact is of supreme importance prognostically.

#### OBSTIPATIONAL OBSTRUCTION.

I wish to speak especially of obstipational obstruction caused by large gall-stones appearing in aged people. These stones formed originally in the gall-bladder, obtain the size of small hen's eggs. Through inflammation they unite themselves with the duodenum and ulcerate into the cavity of the intestine to begin their journey to the anus. The blockade is chronic, the colic periodic with great severity, as the concretion passes onward. These produce the obturational ileus, and unless remembered will puzzle the diagnostician. Two cases have recently come under my personal observation. The stones finally passed, being fully the size of hen's eggs. This type of obstruction does not cause the shock with cold hands, etc., seen in strangulated hernia.

Two points of value diagnostically I would like to mention in intestinal differentiation are:

That in diseases producing pain of a colic-like nature located in the large intestine, the pain as a rule is generally below the navel in the hypogastric region.

On the contrary, when in the small intestine, it appears above the navel, or in the umbilical area.

Again: A point of very great value is that in disease of the *large bowel*, pressure on the abdomen over the site of the colon (which in its transverse division is above the umbilicus, and in its ascending and descending divisions to either side of it) will produce pain in the *mid-abdominal zone* without pain at the point of pressure.

In lesions of the small intestine, the pressure in the area below the navel causes pain in the supra-umbilical zone.

#### CHRONIC PAIN.

Attention should be directed to a consideration of a type of dull, persistent, distressing pain felt over the right hypochondrium, of which patients make great complaint, and which is associated with marked enlargement of the liver, and usually with some increase in the size of the spleen. Three distinct entities have to be considered; for though similar in the location of the pain, they have little in common. I refer to Pick's pericarditic pseudocirrhosis, Hanot's hypertrophic cirrhosis and Banti's disease. All show marked enlargement of the liver.

*Pick's syndrome* can always be detected if a general physical examination is in order. The presence of pulmonary tuberculosis; the adherent pleura and pericardium shown by the absence of Litten's sign, and the retraction of the chest wall with each cardiac pulsation; the presence of polyserositis, and an associated abdominal ascites.

*Hanot's cirrhosis* shows the symmetrically enlarged, hardened liver, enlarged spleen, fluctuating jaundice—*no ascites*—irregular temperature, and a *low systolic blood-pressure*.

*Banti's cirrhosis*—described by Banti in 1894. The enlarged spleen, and liver, the marked anemia with leukemia, the tendency to hemorrhages, the presence of ascites.

With these diseases in mind, it is readily possible to analyze the painful types of splenomegalia associated with secondary anemia or *splenic anemia*. Splenic anemia can hardly be considered a definite clinical entity, as it includes several types. They all, however, produce a discomforting abdominal distress or pain.

The Gaucher type occurs in the young, the anemia is usually of the chlorotic type; no ascites and no enlargement of the liver. Microscopically it has a typical cell—large, vesicular with a small nucleus.

Von Jaksch's pseudoleukemic anemia of infants is not receiving as much attention as formerly, for the reason that it has been determined to be one of the atypical types of blood disease. It occurs in children, and is characterized clinically by enlargement of the spleen, abdominal pain, and secondary anemia. Associated rickets is present.

Hemolytic jaundice, described by Hayem in 1898, shows enlarged spleen and jaundice and tendency to hemorrhages. An important laboratory finding is in the vital staining of the blood. Microcytes and reticulated red cells, microblasts, are found in increased numbers.

Blood examination becomes imperative in the making of abdominal diagnoses. Wright's stain is to be recommended.

#### ADHESIONS.

Highly important is it to understand the relationship of adhesions to abdominal pain. Obscure conditions, proving elusive to diagnosis, can be illumined if the story of adhesions be recalled.

As an end result of nearly all processes both inflammatory and otherwise, in the abdominal cavity, these unions of the visceral and parietal surfaces of the peritoneum can be productive of great distress. The pain is generally localized, and is produced by pressure or tension.

When these adhesions form between the stomach and anterior abdominal wall, the pain is often increased after eating, and a sense of fulness results. Inasmuch as the hollow viscus, namely the stomach and the intestine continually change size and shape, the pull upon the adhesive area with each peristaltic wave produces a very annoying, stubborn, debilitating and exhausting pain.

These adhesions may be divided into groups, thus aiding in the differential of these obscure pains.

*A. Gastric group:* Including cholelithiasis; ulcer of the stomach and duodenum; traumatism of the stomach, liver, pancreas and duodenum; and carcinoma of any of the above mentioned organs.

*B. Intestinal group:* Which is particularly associated with the appendix and sigmoid.

*C. Pelvic group:* Includes lesions of the tubes, ovaries, and uterus.

*D. Peritoneal group:* Including all lesions in which primarily the peritoneum is involved, as in tuberculous peritonitis.

The chronicity of these cases is a valuable point in the diagnosis.

The pain, the pyloric syndrome, the nausea, the vomiting, the tenderness over the epigastrium and hypochondrium—the tendency to colics and loose stools or the reverse, constipation—the abstinence from food—the emaciation, all aid in calling up the picture of adhesions.

In the *gastric* group: Recall that there is no vomiting in duodenal ulcer, and that two symptoms—pain two or three hours after eating, and relief by eating—are extremely suggestive of duodenal ulcer. Their absence negatives the diagnosis of duodenal ulcer, and as the picture rarely wholly simulates gastric ulceration, this may be eliminated.

*Intestinal adhesions:* An interesting case recently came under observation, which after study and X-ray help, was diagnosed as adhesion about the cecum. Operation showed the ileum at its junction with the cecum as completely kinked. The relief of the adhesions checked the pain, vomiting and distress, and permitted of a complete recovery.

*Pelvic adhesion:* May I advise the recalling of this condition in every chronic case of pelvic suppuration, especially in cases of salpingitis, or even after operative childbirth.

Remember, in the words of Dr. Charles L. Mix, "We must learn that it is by no means necessary to diagnose disturbances of the abdomen as diseases with names. The diagnosis is often merely the determination of the anatomic conditions consequent upon healed up disease."

In my judgment, adhesive conditions are responsible for a large group of diagnostic failures, and therefore advise that the thought of adhesions remain uppermost in the practitioner's mind, particularly in chronic cases.

#### SYPHILIS OF THE LIVER.

Syphilis of the liver produces a frequent, and at the same time, one of the most obscure and difficult pains to recognize. This pain, which may be very distressing, is situated in the epigastrium. It is dull and persistent, does not seem to be definitely related to meals,



although in a general way it comes on after eating, especially with a full stomach. It is not relieved by soda or drugs. There is no nausea, no vomiting, or occult blood.

The liver is enlarged, and because of the presence of jaundice or ascites, the diagnosis of cirrhosis of the Laënnec type is made.

The differential hinges on the abdominal findings, viz.: The rounded liver edge, the presence of depressions, irregularly placed over the liver surface, or the finding of deep incisures with a lobulated formation. The liver is hard or resistant. This condition of the liver gives the clue to diagnosis. Should a clue be obtained, the Wassermann reaction will prove the specific origin.

Three types are recognized:

I. Cases resembling cirrhosis of the Laënnec type.

II. Cases resembling malignant tumors.

III. Febrile cases resembling abscess of the liver.

The first must be differentiated by the absence of an alcoholic history; by the presence of the incisures, and the Wassermann reaction, and by the response to mercury and iodides. Also noting that the jaundice is of a lighter grade for the terminal stage found in alcoholic cirrhosis.

*From the malignant tumor* by the Wassermann reaction and by the response to mercury and iodids.

*From abscess* by the failure to obtain a history of amebiasis. It is the acute stage with perihepatitis that simulates abscess.

#### SUPERIOR OF INFERIOR MESENTERIC VENOUS THROMBOSIS.

These conditions occur just often enough to deceive the doctor, and though rare in general practice, should be kept in mind.

Thrombosis of the *mesenteric veins*, in the majority of cases, are diagnosed as intestinal obstruction. The symptoms are also similar to embolism of the mesenteric arteries, though if anything more severe: *Pain*, localized or general, sudden in onset, with nausea and vomiting, abdominal tenderness and signs of obstruction. This pain may be a dull ache, or a *very severe colic*. The cause of the pain is thought to be due to the contraction of the intestine, which makes it analogous to the pain of obstruction, or simulate attacks of intestinal colic from abdominal arteriosclerosis (angina abdominis).

The character of the vomitus depends on the severity of the in-

vovement—first, normal stomach contents; later, bile, fecal matter or blood.

The differential is difficult. The most characteristic signs are sudden onset of colicky abdominal pains with a fall in temperature and passage of blood-stained stools; later, intestinal obstruction and abdominal distension. It is frequently associated with thrombi elsewhere. Differential must rest upon the other signs.

I mention this condition just to awaken your minds to the same.

#### THROMBOSIS OF THE PORTAL VEIN.

Usually caused by some pathological disease in the neighborhood of the vein. The symptoms of pylethrombosis are different in septic and non-septic cases. Septic or suppurative pylephlebitis results in multiple abscesses in the liver. Appendicitis is very frequently the exciting cause. Onset sudden with a violent chill, high fever, profuse sweating and at times *intense pain*; a remitting fever, gastrointestinal symptoms—a typhoid state—collapse and death to close the scene.

#### COURVOISIER'S LAW.

In cases of deep and persistent jaundice this law, also referred to as the Bard—Pick syndrome, should always be kept in mind to aid the differential in cases of *stone* and *carcinoma*. The presence of jaundice, gastric distress, and palpable tumor in the gall-bladder area opens the question of diagnosis. Is it stone or cancer? (New growth) When jaundice exists for a protracted period, a positive closure of the biliary tract is evident.

This law is generally accepted to mean that a jaundice associated with a palpable gall-bladder always indicates malignancy. Unassociated with a palpable gall-bladder, it indicates stone.

The point of location of the neoplasm is most often at the papilla of Vater, or in the head of the pancreas.

Time forbids a further consideration of the causes.

Let me say a few words regarding a vital function test, that I count of great aid in diagnosis, especially when considering the disturbances connected with liver and gall-bladder. I refer to the urobilinogen test, or so-called Ehrlich's aldehyd reaction. The test solution is made by taking four grams of para-di-methyl-amino-

benzaldehyd and adding to it 200 c.c. of a 20 per cent. solution of hydrochloric acid; the resultant liquid is a pale yellow fluid, stable if kept in glass-stoppered bottles. Five or more drops are added to the *fresh* urine. In the presence of urobilinogen a rose red, or cherry color, appears.

Urobilinogen has been found in the urine of many pathological conditions, chief of which are cirrhosis of the liver, cholangitis, infectious diseases, disturbances of the bile function, etc. While questioned by some, the test proves of the greatest value in daily work.

The galactose test of Richard Bauer proves of great value in determining the condition of liver function in catarrhal jaundice. The amount of sugar recovered in catarrhal jaundice is greater than in the cases of obstructive jaundice. The test consists in giving 40 grams of galactose dissolved in 400 c.c. of tea, taken in the morning on an empty stomach. The urine is collected hourly and examined for sugar. The test proves of value in all cases of chronic jaundice to determine the cause of the jaundice.

Finally:

Stomach analysis, blood analysis, Einhorn's duodenal tubes, string tests, Robert's occult blood tests, urinary analysis, stomach dilatation with soda and tartaric acid, radiography with its recently improved technic, careful inspection, and technical palpations, etc., are all vitally essential to the developing of an accurate differentiation in abdominal diseases.

#### THE ARCHIVES OF DIAGNOSIS

Editor, HEINRICH STERN, M.D., LL.D., F.A.C.P., 250 West 73d St., New York  
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# THE ARCHIVES OF DIAGNOSIS

A QUARTERLY JOURNAL DEVOTED TO THE STUDY  
AND THE PROGRESS OF DIAGNOSIS AND PROGNOSIS

Vol. X

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OCTOBER, 1917

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No. 4

FOUNDED AND EDITED BY  
HEINRICH STERN, M.D., LL.D., F.A.C.P.  
New York



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HEINRICH STERN, M.D., LL.D., F.A.C.P.

## Obituary

Dr. Heinrich Stern was born in Frankfurt fifty years ago. After completing a liberal education there, his father, who had suffered greatly from the oppression of autocratic Prussian rule, sent his son to America to escape a similar fate. Soon after arriving here he began the study of medicine and was graduated twenty-two years ago from the St. Louis Medical College. After a few years of general practice he devoted himself exclusively to the study of diseases of metabolism and zealously pursued this course to his very death.

Dr. Stern possessed a rare insight into the needs of the medical profession, and was imbued with an ardent desire for its betterment and for the recognition and advancement of internal medicine, his particular field. To accomplish this object he founded two years ago The American College of Physicians. This organization will be a lasting monument to his memory.

Thirteen years ago he founded the INSTITUTE FOR MEDICAL DIAGNOSIS AND RESEARCH in the City of New York. It anticipated the plans of the Rockefeller Institute by some years, but it perished through lack of financial support.

Medical Organization was always a strong feature with him. He founded and was the permanent Secretary of the Manhattan Clinical Society. Two years later he founded successively the North Side Medical Society and the Manhattan Medical Society. This latter Organization under his guidance became a prominent Medical Organization. Many of the most prominent Physicians and Surgeons of America delivered addresses before it.

In 1905, he was made Chairman of the Section of Therapeutics of the American Medical Association. He was a member of many other Organizations, notably:—

New York State and County Medical Society,  
Harlem Medical Society,  
Medical Society of Greater New York,  
American Urological Society,  
American Public Health Society,  
American Therapeutic Society,  
Fellow of the New York Academy of Medicine,  
Society for the Relief of Widows and Orphans of Medical Men.

Among his colleagues in New York, he was known principally because of his work on DIABETES. Early in his career, he won the prize offered by the New York County Society for researches pertaining to the etiology of diabetes. Shortly before his death, he published an excellent monograph on its treatment. He was author of at least half a dozen other books on Medicine, some of which were published in the German language.

Every year at least a dozen articles on various subjects of medical interest appeared from his pen in the Medical Press of this country and in Europe.

Ten years ago he determined to found a Journal devoted exclusively to Diagnosis, to be the forum from which the greatest diagnosticians in America



should hold forth. It is known as THE ARCHIVES OF DIAGNOSIS, and will be continued as a lasting monument of his achievements to this field.

Owing to his wide acquaintance among the medical men throughout the country, it was possible for him to secure articles from the leaders in the profession, and no journal in this country has had a higher clientele, either among writers or readers of medical literature.

At one time or other he was connected with many Charitable Institutions in the City of New York, among which were the:—

Metropolitan Hospital and Dispensary,

Red Cross Philanthropin, which he founded.

The German West Side Hospital and Dispensary.

At the time of his death he was Visiting Physician to St. Mark's Hospital, Methodist Episcopal Home; Consulting Physician to Seney Hospital, Brooklyn, Central Islip Hospital, The Portchester Union Hospital, Glens Falls Hospital, Union General Hospital of Boston, and White Plains Hospital.

During his time of active work, he was professor of Medicine in the German West Side School of Medicine, and in the School of Medicine of Boston College.

Three years ago he received the honorary degree of LL.D., and for two years before his death he was a member of the Medical Reserve Corps of the United States Army, which office, owing to ill-health, he had to resign.

This many-sided man, with his wide scope of knowledge, left an indelible mark on American Medicine, and his influence will remain for generations. His passionate desire to tell the unvarnished truth naturally netted him some enemies. But he counted innumerable men in all the walks of life among his staunch friends. Through his death the Medical profession loses a leader, and the ARCHIVES a skilled editor.

## PUBLISHER'S ANNOUNCEMENT

During his protracted and fatal illness, the editor of the ARCHIVES OF DIAGNOSIS was not able to finish the October, 1917, issue, and it was impossible to complete it and to get it to our subscribers before this time. We apologize for this long delay, feeling that the readers of the ARCHIVES will appreciate the difficulties which we encountered in the matter. They will find in the October Number the final effort of this able man whose portrait with an obituary notice will be found in the front of this issue.

We are happy to say that Doctor Dwight Clifford Martin, assisted by Doctor Edgar W. White, both of New York City, has very kindly consented to undertake the arduous task of the Editorial work. We do not hesitate to think that he will continue the work which Doctor Heinrich Stern began and carried out so brilliantly and successfully, to the full satisfaction of the subscribers of this excellent periodical, and feel confident that he will meet the expectations of those interested in the welfare of the ARCHIVES OF DIAGNOSIS, and be a worthy successor to the late lamented Editor.

As the October issue, 1917, has been so much delayed, we have thought it best to omit the January and April Numbers for 1918 and start Volume XI with the July issue, 1918, thus making the subscription to run from July, 1918, to July, 1919. We sincerely trust that our readers will fall in with this idea, as it cannot prove detrimental to their interests; for whatever subscriptions (1918) have already been paid will be credited for the four issues that are to belong to Volume XI.

We also venture to hope that the subscribers to the ARCHIVES will continue their faithful support as they have done in the past. It will be our aim to bring out in future all issues as close to the first of the month in which they fall due as is compatible with the manufacturing of the Journal. We tried our best in the past to come up to this standard, but the difficulties were insurmountable, and we had to content ourselves with belated issues. We feel

that this must have been quite a trial to the readers, and we take this opportunity to thank them for the leniency and kindness they have shown in the past in this direction.

Assuring them that we shall do everything in our power to remedy this evil and promising that the July, 1918, issue will be ready on that date, we shall begin Volume XI under more propitious auspices and thus lay a valid claim to the uninterrupted patronage of our subscribers.

New York City, April 1, 1918.

REBMAN COMPANY.

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**Special Articles**

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DIAGNOSTIC VALUE OF THE DUODENAL TUBE AND  
X-RAY FINDINGS IN THE ULCER OF STOMACH  
AND DUODENUM

By CARL BECK

Chicago

The only positive proof of the presence of a duodenal ulcer or ulcer of the stomach can be gained by the inspection through laparotomy, and only through this method of positive diagnosis it has been established that the ulcer is much more frequent than it was supposed to be. As long as we had to depend in our diagnosis on some of the obscure symptoms like occult bleeding or history of pain in relation to feeding or else on a profuse bleeding from intestine or stomach when the patient was in a dangerous condition, the diagnosis before the operation was rather hazy. Pain at certain intervals and in certain relations to food ingestion, local tenderness, microscopic or occult hemorrhage were the most common of symptoms, but relying on them the surgeon was often disappointed by finding either another pathological condition which sometimes did not even require surgical interference or else not finding anything at all.

The X-ray has brought, like in many fields, a great deal of relief. The outline of the stomach in the pictures and especially that part of the stomach which is the common seat of ulcers, the pylorus and post pyloric region and further the small curvature have shown niches and distortions which indicated clearly the seat of ulcera-

tions. Better and more conclusive than that was the condition of the stomach during its action as observed by the fluoroscopist showing certain abnormal contractions in cases of ulcers so that fluoroscopy and skiagraphy combined have added a great deal to the diagnosis of ulcerated conditions.

Still ulcers are not all of the same character and a very shallow abrasion without any infiltration of its base will give an entirely different picture than the infiltrated punched-out ulcer of the stomach. In this latter case one will find the little button of bismuth which indicated clearly the loss of substance while in the superficial ulcer without any infiltration such will be totally absent. It may be even an exulceration which bleeds very profusely, but has no infiltration and, therefore, there will be absolutely no trace of it neither in the picture nor in the fluoroscopic examination. It may also escape our observation during the operation under such conditions as it will not be palpable from the outside and still it may be a very extensive defect in the mucous membrane. In such instances only the opening of that part of the intestine will reveal the defect and the bleeding surface.

I have had a case of a bleeding ulcer in which the patient has almost bled to death from a very superficial ulcer near the pylorus and in which I sutured this ulcer by a through and through suture and thus saved the patient, and if it had not been for the extensive hemorrhage from the stomach, I should not have been inclined to open the stomach, because the palpation of all the parts with my fingers showed almost normal structure.

Among other observations, I have had a case in which after a gastroenterostomy the patient continued to vomit blood for several days and finally I was induced, by the seriousness of his condition, to reopen the stomach and examine the suture of the gastroenterostomy which was perfect, to find that a very small point-like ulcer on the small curvature containing a tiny artery was the cause of the bleeding. No external examination did help nor did the gastroenterostomy change any of the pathologic conditions existing.

A very reliable method is the duodenal tube which is used little in the average practice of surgeons, but gives especially in the case of duodenal ulcerations valuable information. The instrument is well known. Its use is very simple. The patient learns to swallow

the instrument and it gradually passes into the duodenum and we are able to withdraw with our syringe fluid of the duodenum, and by careful examination, we find blood in the form of small particles coagulated or even in the form of tinged bile microscopically and chemically in the contents of the duodenum. Under normal conditions such bile does not contain blood, but by continuous and persistent appearance of blood, we will be able to find this a very valuable method for diagnosis. The swallowing of a thread is a valuable method, but it is only in those cases of ulcer in which the thread touches the same as it is, it sometimes passes these ulcerated portions and does not touch the ulcer. A thread forms a line and if this line touches that ulcer, it may rub off some of this ulcerated surface and stain the thread, while the contents of the duodenum especially at certain times of the day contaminated with blood specks are almost a positive proof of a duodenal ulcer. The only objection that could be made is that such blood is the result of traumatism, but we find also in the examination a proof that it is often old blood disintegrated already and coagulated, showing that it has been in the duodenum for some time, a few hours at least, when it has been withdrawn from the duodenum. In eroded large ulcers we do not use this. If we have made the diagnosis by other methods more or less positive, we do not use it, but in cases of doubtful duodenal ulcer it is especially welcome.

One of our associates, Dr. Leopold Frankel, has made a particular study of this class of cases and will perhaps write at some future time a detailed article on this method of diagnosis.

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## THE DIFFERENTIAL DIAGNOSIS OF CHRONIC DUODENAL AND GALL BLADDER DISEASE.

By JACOB GUTMAN  
Brooklyn-New York

The difficulties and pitfalls in arriving at a correct diagnosis of diseased subphrenic organs are admitted not only by surgeons, but even by the most skillful internists as well. Mistaken diagnoses are especially frequent in the region of the right hypochondrium. This is because the possible sites of disease, combinations and varieties of

pathological process are more numerous in this quadrant than in any other. We have become quite accustomed to have our errors made in this region revealed by operation or post-mortem, and readily console ourselves rather than have qualms of conscience.

Of abdominal disturbances, the affections of the gall bladder and duodenum constitute no insignificant chapter. With the exception of the appendix, which, during the past decade, has been abused most pitilessly, and whose persecution has lately somewhat relaxed, the duodenum and gall bladder constitute fertile fields for the surgeon and physician. Here infections, ulcerations and new growths are not rarities and the differential diagnosis of such abnormal conditions is frequently in demand.

With the object of stimulating greater efforts toward correctness in diagnoses, this paper will briefly review some of the means at our command, which must be utilized with a fair degree of accuracy in order to *do justice* to the difficult subject of differential diagnosis. The interpretation of symptoms will be limited to those characteristic of the chronic diseases of the gall bladder and duodenum, the acute inflammatory or accidental cases not being within its scope. Neither will carcinoma of the gall bladder, a subject very extensive in itself, receive consideration here. The differentiation in the diagnosis will apply here only to chronic ulcer, with and without stenosis, chronic gall bladder inflammation, cholelithiasis and affections simulating these.

*Symptomatology.* We divine the first clue to a diagnosis when the patient relates his physical sufferings. It would be proper, therefore, first of all, to consider the value of the complaints related and their interpretation as factors in differential diagnosis. Some of the symptoms enumerated will be found quite characteristic, if not typical, of gall bladder disease, while others may be more expressive of a duodenal condition. Such symptoms are very valuable. But, on the other hand, the symptoms related may be common to *both* conditions. These are of lesser value in differential diagnosis.

It is beyond the scope of this paper to go into great details of every symptom encountered; therefore only the most important and most dependable will be considered here.

It is quite true that a number of pathological gall bladders and

duodena exist for many years without producing symptoms. It is, nevertheless, seldom that the period of quiescence may last the lifetime of the individual. As in other diseases, such as tuberculosis, where at necropsy diseased foci are frequently revealed which gave no signs during life, so also in gall bladder and duodenal disease we meet with cases giving no trouble whatsoever for many years. Quiescent gall stones have been found by Murat, for example, in as many as 20 per cent. of his cases, although duodenal cases of this type are less frequent.

The symptoms of duodenal disease vary; the position, size, depth, number and extent of ulcerations, the presence or absence of induration and stenosis, the constitutional habitus, the age and occupation, the state of the adjoining organs closely related anatomically or physiologically—all these determine the character of the symptomatology. There is no syndrome of symptoms absolutely pathognomonic of either gall bladder or duodenal disease characterizing in other infections and diseases. Each and every symptom, whether subjective or observed by the physician, has to be taken into consideration, interpreted and judged upon its merits in the differential diagnosis of gall bladder and duodenal disease.

The following symptoms deserve consideration:

1. *Pain* (subjective) in *duodenal disease* occurs usually periodically with irregular intervals, generally inconstant, mostly late (from two to five hours) after a meal, frequently at night, not always following ingestion of unusual food. It is mostly felt below the xiphoid, or to the right near the border of the rectus muscle; and is often confined to this point, although it more frequently extends downward toward the thigh or backwards to the spine. A point of tenderness is to be found dorsally just below the right twelfth rib, near the spinous process of the same vertebra. In front, the pain may be elicited by pressing a point about one inch below the gall bladder notch or midway between it and the median line (Boas). The character of duodenal pain is dull to boring or cutting, and is often relieved by ingestion of food, "hunger pain," also frequently by pressure and change in posture.

In *gall-bladder* inflammation, whether complicated by the presence of stones or not, the pain is of a somewhat different character. Here the pain is felt mostly in the right hypochondrium, either at



the gall bladder notch, or to the right of the above described duodenal point. In its radiation it differs from that of duodenal disease by shooting upwards toward the right shoulder, or to the right, along the liver border, and not downward or through to the spine. It has none of the characteristics of "hunger pains" and is not relieved by the intake of food; on the contrary, emptying the stomach by vomiting relieves the pain; nor do alkalies or spices exert their peculiar effects as in duodenal colics. The attacks in gall bladder cases bear no special relationship to meals, posture or to time of day. The dorsal point of tenderness is more to the right of that in duodenal cases, nearer to the spine, being at the transverse process of the last dorsal vertebra.

2. *Tenderness and resistance* (defense musculaire), if properly and carefully elicited, are of great diagnostic value. No matter how chronic the process or mild the case, very careful depression at various points of the abdominal wall with either finger or rubber-tipped pencil will reveal the most sensitive spot and thus the diseased area. The involuntary muscular resistance, if elicited, is invaluable, but its detection and interpretation greatly depend upon the personal equation of the examiner. The sensitiveness of certain Head zones also help to differentiate a gall bladder condition from pyloric and duodenal.

3. *Periodicity* is characteristic of duodenal attacks; less so of gall bladder. The intervals in duodenal cases are of varied length, extending even into years, and are usually remarkably free from gastro-intestinal disturbances. Frequently, however, symptomatology of the formerly so familiar types of nervous dyspepsia and neurasthenia are concomitants of chronic duodenal ulcer in the intervals. The attacks proper come on suddenly, unexpectedly, often like a bolt from a clear sky, perhaps during the night, and seemingly without any provocation. On closer investigation though such cases as an indiscretion in diet, a cold, exhaustion, acute mental strain or depression may be identified with the attacks. Moynihan lays much stress upon some such provocative cause as mentioned above. In many cases under his observation the writer was enabled to trace the origin of numerous attacks to definite causes, varying greatly in their character, from the ingestion of sauerkraut to the act of copulation.

In gall-bladder affections, on the other hand, with the exception of the typical gall stone colics, periodicity of attacks is not as customary as in duodenal disease. Nor are the intervals entirely free from gastro-intestinal symptoms; heartburn, nausea, vomiting, epigastric fullness, water-brashes, etc., constitute the sufferings of the patients during intervals.

*Vomiting* is not as frequent an occurrence in duodenal disease as in gall bladder. Oppenheimer claims that vomiting occurs only in about 17 per cent. of proved cases of *ulcus duodeni*, while in gall bladder cases the percentage runs up to above 50 per cent. Exception must here be taken to those cases of old, indurated, callous ulcers, in which stenosis and gastrectasia are a common rule. The vomiting, if present, is then generally of a golden yellow color, except in intrapapillary stenosis, while that of gall bladder disease is usually greenish and bilious. Vomiting in duodenal cases is a late symptom, and is associated with gastric dilatation, pylorospasm and chronic dyspepsia. In gall bladder disease vomiting is an earlier manifestation.

*Gastric Peristalsis*, observed extra-abdominally in the pyloric region, especially if of the reversed type, is of great value in the diagnosis of pylorus hypertrophy and stenosis. This symptom points to the presence of duodenal disease, particularly so if stenosis pylori carcinomatosa had been excluded.

*Gastric Analysis.* A chemical analysis of the gastric contents after a test meal is another helping factor in the differential diagnosis between gall-bladder and duodenal disease. Hyperacidity was found by Ohly in only 18 out of 77 gall-bladder cases; in 46 HCl. was either lacking or invariably deficient; and in 13 normal secretory findings were obtained. Moynihan claims a normal gastric analysis to be the rule in the majority of duodenal cases. However, hypersecretion and hyperacidity with pylorospasm are regularly found. Gastric motor insufficiency is quite common in duodenal cases, with delay in the emptying power of the organ. Too much stress must not be paid upon the acidity of the gastric contents. Extremes of acidity have been encountered by the writer in duodenal as well as in gall-bladder cases. Greater reliance may be placed upon some of the other findings of the extracted meal. The presence of blood, for example, is more characteristic of duodenal disease, while the ad-

mixture of bile is more common in gall-bladder cases. Blood in an extracted oil breakfast (Boldyreff and Volhardt) points to duodenal disease. Occult blood in the stools collected after several days of blood-free dieting, if open bleeding points of the low portion of the gut are excluded, speaks for fresh duodenal lesions. Moynihan claims *pronounced* hemorrhages to occur in 38 per cent. of duodenal lesions, while in 50 per cent. minor oozing is found upon frequent occasions. Melena is, of course, of decisive importance in perforated ulcers.

*The Einhorn thread or bucket test* has been found by some useful as a diagnostic method, although other not less painstaking investigators place less reliance upon the test. If the technic employed is the correct one, the test is of undoubted value, and in a limited number of cases employed has so served the author.

*The Blood* of the individual may also be utilized in the differential diagnosis of the two conditions. Friedman described the occurrence of hypercythemia in 83 per cent. of his duodenal cases with absence of large mononuclears, also eosinophiles, and the presence of a relative lymphocytosis. These findings have been verified by several other investigators. While such a blood picture is not entirely diagnostic of duodenal disease, the author having encountered similar blood states in bronchiectasis, chronic tuberculosis, dysthyroidism, intestinal autointoxication, etc., it is, nevertheless, of help in differentiating duodenal from gall bladder disease, as in the latter condition no such blood states have ever been observed.

*Hypercholesterinemia* is claimed by Henes to exist invariably in all gall stone conditions. In a summary of 21 cases, he says: "A hypercholesterinemia seems to be the fundamental primary etiologic factor in the formation of gall stones"—and—"in cases of cholelithiasis without fever a hypercholesterinemia is invariably found"—and he further states, that "conditions simulating cholelithiasis can be differentiated therefrom by a cholesterin-serum determination. Particularly does this apply in the differentiation of *ulcus duodeni* from cholelithiasis." Whether Henes's contention is applicable in every instance of gall bladder disease is debatable. Several physiological chemists whom the author has interpreted do not seem to share this view. Nevertheless, it is a matter of fact that the blood of individuals affected with gall stones usually contains an excess

of cholesterin. This excess is certainly more commonly encountered in cholelithiasis than in duodenal ulcer. We are, therefore, justified in applying this test also in the differential diagnosis of the two mentioned conditions.

*The examination of the stomach* frequently helps in the differentiation of gall bladder from duodenal affections. While in cholecystitis the stomach is but little affected in its size or position, in duodenal disease the organ is frequently enlarged, the greater curvature is much lower than normal, the pylorus is located to the right of the median line, the antrum dilated, peristalsis exaggerated, the emptying time increased.

*The constitutional habitus* of the individual affected is quite different in the two conditions. Gall-bladder disease is met with mostly in females, the obese, sedate, in sluggish individuals, or in those whom Fraenkel classifies as thyrotrope; while duodenal disease is more common in males, of moderate weight, nervous temperament, vagotonics.

*Etiological data.* A diligent search for the possible etiological factor must be instituted in every question of differential diagnosis. It is necessary to take a complete history: "Qui bene interrogat, bene diagnoscit." The etiological factor plays no minor rôle in differential diagnosis. Although no definite conclusions have as yet been reached regarding the etiology of either gall-bladder or duodenal disease, certain facts have so frequently been associated with either of these conditions that they deserve more attention than if mere coincidences. Chronic alcoholism and nicotinism, for instance, have been advocated as a cause of duodenal ulceration by such clinicians as Boas, Ewald and Albu. Kehr identifies duodenal disease with traumatism. Schmieden blames an abnormal position of the stomach causing sharper angulation of the duodenum resulting in protracted retention of the chyme and of increased acidity within the bulbous as the cause of ulceration. Eiselsberg, Payr, Dietrich and others ascribe post-operative capillary thrombosis to be the origin of a number of ulcers in post-operative cases. Pous speaks of other vascular causes occurring in pathological states of the heart and blood vessels. Perry considers nephritis as productive of intestinal ulceration. Claude mentions tuberculosis as an important factor; Rosenou, a specially cultivated streptococcus; Friedman

hyperadrenalism. The theory of self-digestion, which has prevailed for some time, seems to be refuted by such men as Moynihan, Chvosteck, Robson and Mayo.

From the great number of etiological possibilities enumerated, it would seem that the etiology of a case in question could not be utilized for purposes of differential diagnoses. This assumption, though, is not altogether justified, for when we analyze the characteristics of the etiological factors, it becomes possible to help make deductions from them. The occurrence of gall-bladder disease in females, at the age of 40 or over, in those inclined to obesity and who have passed one or more pregnancies, or who have had some general or local infection, in individuals affected with constipation, in great eaters, etc., will permit classifying the case into the gall-bladder division, while the occurrence of a case in a male, below the forties, rather slim and lively, who uses tobacco, indulges in alcohol, and loves spicy food, a hard worker and great hustler, etc., justifies us in suspecting the case to be a duodenal one rather than a gall-bladder one.

*X-ray examination.* Last, but by far not the least, a most important method of differential diagnosis is the radiographic examination. Extremely valuable and definite information is obtained by this method. The author has made it a rule to make no final diagnosis in chronic abdominal cases unless the patient had been given the benefit of an X-ray examination in addition to all other practical clinical tests. If the radiogram shows unquestionably gall stone shadows, or the outlines of an enlarged gall bladder, the question of diagnosis is certainly eliminated. But this is uncommon. While it is true that the technic of photographing gall stones has lately been given special attention, it has as yet not reached the pinnacle of perfection. It must be admitted, though, that the latest improvements in photographic plates, apparatus, exposures, positions, etc., have increased the number of successful gall stone radiographs.

Pfahler, George, Case, Hirsch and others have shown the possibility of exhibiting gall stones on plates in more than 60 per cent.; other even claim as high a percentage as 90. Improved technic enabled Case to reproduce stones containing only 0.4 per cent. of calcium oxide, a very insignificant proportion of mineral. Pfahler and Case claim the diagnosis of even pure cholesterin stones under

favorable surroundings. There is danger, though, in the interpretation of gall stone shadows. Caution must be exercised not to mistake calcified lymph nodes, cartilaginous calcium deposits or a renal calculus for gall stones. A close study of plates has often enabled the writer to detect unexpectedly stones in radiograms of the right hypochondrium taken altogether for other purposes. Nevertheless, the direct method of gall-bladder photography is still a difficult procedure, but not always an indispensable one; the indirect, in our opinion, is as valuable, if not more so. It helps the diagnosis of duodenal, appendical, gastric and intestinal disease, and thus leads to the diagnosis of gall-bladder disease per exclusionem, especially if complicated by adhesions to surrounding structures, an occurrence not uncommon in gall-bladder disease.

As to the X-ray examination in duodenal disease, even more valuable data are thereby obtained. Haudeck niche; a duodenal residue; a persistent defect of the antrum; spasmodic contraction of the latter; a gastric residue, hyperperistalsis, delayed gastric motility; intestinal hypermotility; tenderness of the duodenal portion if pressed upon during fluoroscopy, etc., help to establish the diagnosis of duodenal disease. Much reliance may be placed upon the radiologic examination, provided perfect technic had been employed and caution exercised in the interpretation of findings, conditions demanding more or less expertness upon the part of the examiner.

*Conclusions.* As may be seen from the above, there are a number of methods at our command in the differential diagnosis of gall-bladder and duodenal disease. A diagnosis should not be based upon any one symptom or method of examination, but upon a compilation of all. An examination by every means is imperative in order to arrive at a correct conclusion. Especially is this true in the complicated cases, where gall-bladder and duodenal disease co-exist, or when either of these are accompaniments of chronic appendicitis, adhesions, new growths, displacements, etc. It is here where skill is needed. But persistence, patience, perseverance and a modern equipment will help to solve the riddle. The surgeon solves the problem with his scalpel as Alexander the Great unraveled the Gordian knot with his sword. A liberal incision saves him strenuous efforts and deliberation. But what as to the consequences to the patient? Is the case really a surgical one necessi-

tating such procedures, or is it one in which the internist should interest himself and correct the infirmity in nature's own way? The author has no eulogies for the surgeon who sees in his scalpel a cure for every ill flesh is heir to. He fails to see the rationale or benefits of surgical procedure so exotic as the resection of a number of yards of gut to cure an arthritis. To the surgeon, however, all credit is due for the manner of handling his cases and the attention he pays to the minutest detail, from the moment of the first incision to the completion of post-operative treatment. Not so always with the physician. Were the physician always as careful, painstaking, as persistent and as thorough in his attention to a case as is the average surgeon, a great deal of surgery and post-operative misery could be avoided. It is well, therefore, to give more attention to the patient, exercise greater care and make greater efforts in diagnosis, use more extensively our modern diagnostic facilities and seek persistently, honestly and energetically the cause of the diseased condition.

Mistakes are often made because we leave many stones unturned in reaching a conclusion. This is not scientific practice. Every effort should be made to take advantage of every possibility, no matter how painstaking and time-consuming it may be, to come to correct conclusions. Otherwise we are not doing the best by our patients or ourselves. While we must honestly admit that in some instances conditions may be such as to baffle even the most earnest and most careful investigator, it is also true that a great number of faulty diagnoses are made, not because of insurmountable difficulties, but rather because of haste in railroading the patient to the operating table, a procedure much simpler. Furthermore, careless examinations, lack of appropriate laboratory facilities, and, what is even worse, cock-suredness of the physician or surgeon in charge of the case are contributory factors in faulty diagnoses. It is deplorable when so many exploratory laparotomies are made with enormous incisions prepared for all probabilities, simply because no serious attempt is made beforehand to ascertain and localize the diseased area. With better pre-operative attention and greater efforts, a good deal of post-operative invalidism could be avoided.

## THE SYNDROME OF CONUS MEDULLARIS AND CAUDA EQUINA APROPOS OF A CASE OF HEMI-LESION

By ALFRED GORDON  
Philadelphia

Lesions strictly confined to the lowest segment of the spinal cord are not frequently observed. The modern war, however, has increased the number of such cases because of the explosives and shells which reach the spinal column and cord either posteriorly or through the abdominal wall. The literature is now abundant with examples of this kind. Apart from the traumatic cases affections of the conus medullaris or of the cauda equina or of both are quite rare. Still less frequently do we observe hemisyndrome of this portion of the central nervous system.

As it is well known syphilis may affect the nervous system in a great variety of forms, so that it may simulate almost any well established affection of the brain or spinal cord. Nevertheless syphilitic lesions confined unilaterally to the conus and cauda are comparatively rare. The following case therefore presents an unusual interest from this standpoint.

L. F., aged 38, business man, contracted syphilis at 20. Eighteen months before coming under my observation he developed the following symptoms: After a hard days' work, associated with much walking, he felt a numbness in the lower limbs and difficulty in micturition. After a two days' rest the numbness improved but the micturition remained unaltered. Soon he commenced to complain of pain in the right leg from the knee down. The pain was very severe. Instead of retention he rapidly developed incontinence of urine and obstinate constipation. The walking became difficult, and he was compelled to give up his work.

At the time of my examination his condition was as follows: The gait is difficult because of the foot drop on the right. It is also in the position of equino-varus. The flexing of the right thigh on the pelvis and of the right leg on the thigh is not difficult while the patient is in bed, but he does it with difficulty while standing or walking. The left lower extremity is normal. It is difficult for him to rise from a seat or go upstairs. The knee-jerk on the right is greatly diminished and Achilles reflex lost. Both reflexes are normal



on the left. Toe phenomenon is absent on both sides. The right leg and foot are cyanosed, cold to touch and there is an ulceration near the external malleolus of three months standing which shows no tendency to heal up. The muscles of the right lower extremity and gluteal region are flabby, diminished in size. Reactions of degeneration are present in the right gluteal muscles, but not in

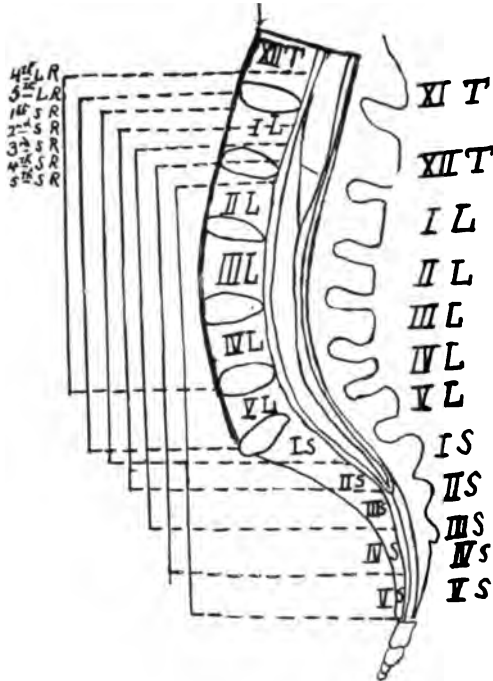


FIG. II. (AFTER RAYMOND)

those of the right lower limb where only a diminution of contractility to both currents is observed.

*Sensations.* (Fig. I.) The pain which the patient experienced at the beginning has totally disappeared, but he feels a numbness in the sole of the right foot; the ground is not solidly felt by him. On the left the sensation is normal. The test for objective sensory disturbances presents the following findings: The *touch* sense is normal. Pain and temperature senses are abolished on the right

side of the anus, perineum, scrotum, penis, internal and posterior surfaces of the thigh down to the popliteal region also on the lower half of the gluteal region. On the scrotal region and penis heat is taken for cold, but cold is not felt at all. The dorsum of the foot is also thermoanesthetic, but pain sense is preserved. Incontinence of urine and feces is quite pronounced. A finger introduced into the anus feels but a very slight contraction of the sphincter. A catheter introduced into the urethra shows almost complete anesthesia. The patient is impotent. Very occasionally there is erection but after the latter has subsided a slow seminal discharge follows without the usual sensation.

An analysis of these symptoms leads to the following deductions: Patient has great difficulty on the right side in rising from a seat and in going upstairs, the right gluteus maximus is therefore involved (small sciatic nerve). The other gluteal muscles are apparently not at all or very little affected, as abduction and circumduction of the right thigh are preserved (the gluteal nerves are therefore not involved). As flexion of the right hip-joint is possible, the right psoas and iliacus muscles are not involved, the lumbar nerves for the first and the anterior crural nerve for the second are consequently intact. Crossing of the right leg is possible; the pectineus, all adductor muscles (the obturator nerve and the great sciatic nerve for the adductor magnus) are therefore not involved. The extensors of the knee (vasti and rectus muscles) and the anterior crural nerve are not involved. As it was mentioned above the patient can flex his knee while in bed, but he cannot do it easily while walking. In bed the flexion of the thigh (which is not disturbed) is sufficient to permit the weight of the foot to flex the knee. The flexors of the knee (biceps, semitendinosus and semimembranosus) and consequently the great sciatic nerve are therefore involved to a considerable extent. The gastrocnemius and soleus (internal popliteal nerve) are involved as the foot is in equinovarus, the tibialis anticus and consequently the anterior tibial nerve are involved (the action of the peroneus longus is antagonistic to that of the tibialis anticus); and as a paralysis of the peroneus brevis leads also to talipes varus, it is supposed that the musculo-cutaneous nerve is also involved. The condition of the toes finds its explanation in the paralysis of the extensor longus pollicis. This detailed analysis

of the condition of the muscles of the right lower limb shows that the following nerves are disturbed in their function; small sciatic (gluteus maximus muscle), great sciatic nerve (flexor muscles of the knee) and various branches of the latter. The motor disturbances are therefore in the domain of the sacral plexus, which is formed by the fifth lumbar and four sacral nerves.

If we turn our attention to the sensory disturbances we observe that the right side of the perineum, and of the anus, the penis, right side of the scrotum and the mucous membrane of the urethra are all analgesic and thermoanesthetic, which is indicative of an involvement of the pudic nerve which again is a branch of the sacral plexus. The condition of the bladder, rectum and anus shows an involvement of the accelerator urinae, compressor urethrae, external sphincter and levator ani; these muscles are all supplied by the muscular branches of the pudic nerve. The patient's sexual power was considerably diminished since the onset of the disease, and ejaculation is involuntary, and not felt by him. The erector penis is supplied by the pudic nerve. The sensory disturbances of the lower extremities are also disturbed over the domain of the sacral plexus.

When an attempt is made to localize the lesion in the present case, one must take into consideration the anatomic arrangement of the sacral plexus and its relation to the spinal cord itself. The sacral plexus is formed by the fifth lumbar and the first four sacral nerves. Figs. II-III. A glance at the diagram will show that the origin of these nerves corresponds to a portion of the cord which is situated between the lower portion of the 12th thoracic and the middle portion of the 2nd lumbar vertebrae. The very lowest portion of this segment is called *conus medullaris*. It comprises the last three sacral segments and the coccygeal segment. The pointed end of the conus continues in the form of *filum terminale* which together with the surrounding nerves descending from the lumbar and sacral segments of the cord called *cauda equina* fill the lower end of the spinal canal from the third lumbar vertebra down to the coccyx. It is evident that a lesion affecting the cauda at the level of the conus may involve also the cord itself and this is the reason why the symptomatology in such cases is complex. A lesion must be in that portion of the cauda which lies below the lower end of

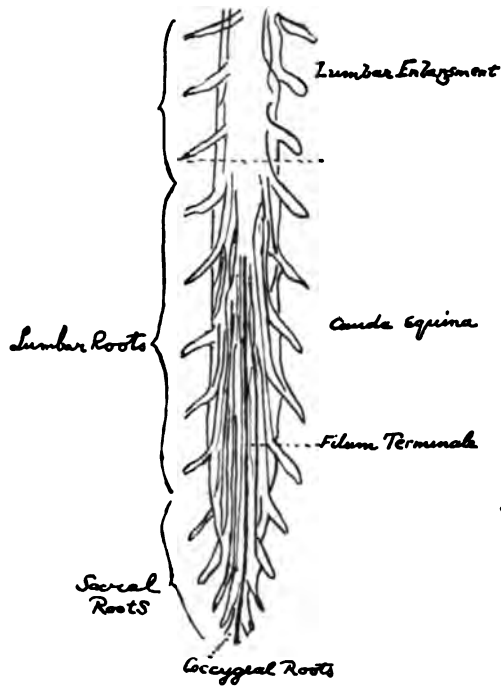


FIG. III

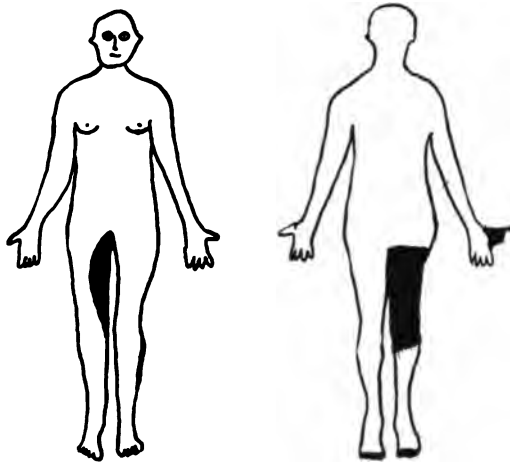


FIG. I. ANALGESIA AND THERMOANESTHESIA

the conus, if a clear case of disease of the cauda is to be made out. A disease of the cauda above that point presents almost always symptoms of a mixed character: those of sacral and lumbar plexuses and of the spinal cord.

From a clinical standpoint therefore several varieties may be observed. 1st. There may be cases, such as tumors of the filum terminale producing eccentric compression upon the nerves distributed in the bladder and rectum. The symptom-group in such cases will be reduced to a paralysis of one or the other viscus. 2nd. In other cases the vesicorectal paralysis may be associated with an anesthesia of the penis and scrotum in men, or the labia majora in women, of perineum, anus, the lower gluteal portion, the postero-superior portion of the thighs; there is frequently also loss of genital function in man. In this group of cases the lesion lies in the conus and the roots emanating from the latter suffer simultaneously. In a 3rd group of cases there may be an association of the above symptoms with sensory-motor disturbances in the lower extremities, and precisely from the mode of distribution of the latter that the topographical diagnosis can be figured out in any given case.

In applying these anatomo-physiological data to the present case one is enabled to place it in one of the categories enumerated above. We find here a combination of the symptoms referable to a lesion in the conus and of symptoms referable to a disorder in the roots emanating from the conus. On the other hand a compressive or an irritative extra-spinal lesion of the roots after they leave the conus may give a similar syndrome. A differential diagnosis is therefore essential. But for better elucidation of this particular feature of the subject it appears opportune to first present the further course and termination of the disease in my patient.

As he first came under my observation in the presalvarsan era he was placed on mercury and iodids. Later on he received several injections of neosalvarsanized serum by the intraspinal route. A prolonged course of massage and systematic exercises improved the condition greatly. He was able to move about and finally discarded crutches and cane. Flexion of the leg improved and finally he began to move the toes and ankle. The ulceration healed up. The most persistent condition was the incontinence of urine and feces. It is only last year that some improvement was noticed in

the control of the rectum. The patient commenced to feel a fullness near the anus which gave him the signal for a bowel movement. This condition remained unaltered. The analgesia and thermoanesthesia remained. The greatest amount of improvement was observed in the motor power. He derived great encouragement from the latter and he soon returned to his former occupation. Six years later he contracted pneumonia and died. Autopsy was not permitted.

In making a differential diagnosis one must consider a nuclear lesion of the conus and a radicular lesion. A priori we must admit that in cases of destructive lesions of the cord the symptoms must become progressively more pronounced and that the opposite is observed in cases with lesions of the cauda. In a case published by Schultze the diagnosis was made by Erb of a disease of the cauda equina, but at necropsy an intraspinal lesion was found. Schultze, himself, observed a case of traumatic lesion of the cauda which ended fatally in a few weeks. It is evident therefore that a progressive or retrogressive course of the condition cannot serve as an important differential element.

The presence or absence of pain has been considered as a diagnostic guide. Pain in the sacral region radiating in the lower extremities is usually indicative of a radicular lesion, but according to Schiff transitory pain is possible in diseases of the conus, although total absence of painful phenomena militates against a radicular lesion. (*Ztschr. f. Klin. Medicin* 1896, p. 87). In Westphal's case (*Charité-Annalen* 1876, t. I, p. 421) there was a radicular lesion, although during life there was a clear-cut picture of disease of the conus.

The state of sensibility has been considered by some writers as a differential sign. They claim that the dissociated character of the anesthesia is indicative of a nuclear rather than of a radicular lesion. (See Higier. *Deutsche Ztschr. f. Nervenheilkunde* 1896, t. IX, p. 185). But sensory dissociation may be observed in other affections of the nervous system, such as Polyneuritis for example.

These few considerations concerning the symptomatology leads to the conclusion that there are no pathognomonic differential elements which could be relied upon as infallible in nuclear affections of the conus and in radicular lesions at the same level. A close reading of the records taken by careful observers shows that the

majority of symptoms referred to the function of the cauda itself are met also in cases described in diseases of the conus. Moreover, pathological records show that whenever the lesion is not strictly limited to that portion of the cauda which surrounds the filum terminale but extends to its upper portion, namely to that which surrounds the conus, the diseased process affects the cauda as well as the cord.

Lesions of the conus and cauda at various levels will present a large variety in their chief manifestations. The records collected in the present war demonstrate several possibilities of injury to the lowest portion of the lumbar enlargement and the cauda equina. Thus we may have an upper or lumbar type, a lumbosacral type, an inferior sacral type and an isolated cauda type. The symptomatology will vary according to the precise localization of the lesion. What especially characterizes all these forms is the disturbance of the sphincters and of the genital functions. In the lower sacral type we observe in addition to the above symptoms also anesthetics in ano-perineo-genital region. The distribution of the disturbances in the motor sphere, in the reflexes, in sensations on the gluteal region and on the limbs, the distribution of pain, the seat of amyotrophic disorders—are all phenomena in relation to the particular area or muscles involved and therefore in relation to the nerves supplying these parts. If the latter are correctly determined, the segments of the cord from which they originate are easily figured out. While the types mentioned are distinct, nevertheless we meet also with intermediary cases.

The present war has also supplied examples of hemi-involvement of the conus medullaris and cauda equina. Here we find the motor and objective sensory disturbances confined to one side. The sphincters are always involved and persist during many months. The reflexes are also disturbed on one side. Irritation of the anus on the affected side does not produce contraction of the sphincter (J. Lhermitte. *Progrès Médical* No. 32, 1917, p. 267). Outside of cases due to trauma, hemi-manifestations of the conus and cauda are not frequently observed. To the latter group belongs my case described above. The initial symptoms, the mode of their development, the character of the sensory and motor manifestations, the trophic disturbances—are all strong indications of a simultaneous involvement of the nuclear and radicular portions of the conus medullaris. As

to the nature of the affection, it is probably a syphilitic meningomyelitis of the conus and of its roots.

The prognosis in the isolated affections of the conus and in those of the cauda equina must naturally differ. As much as cord involvement appears deceiving and is even hopeless, as much affections of the cauda equina always present a hopeful picture. The reason of this capital difference lies in the regenerative processes in the cord tissue and in nerve-roots. In the latter the process is perservering and progressive, in the former it is insufficient and irregular (see G. Roussy. *Bulletin de l'Académie de Médecine* Nov. 1915).

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## THE PROGNOSIS OF HEART DISEASE IN CHILDHOOD

By F. L. WACHENHEIM, M.D.

Attending Pediatricist to Sydenham Hospital  
New York

There is at hand very little literature concerning the prognosis of heart disease in childhood. The subject is nevertheless, of the greatest importance and interest, ranking a close second to the prevention of tuberculosis at the same period of life, as from three to five per cent. of all school children suffer from some form of cardiac disease.

We may divide our subject into the following headings, namely, congenital heart defects, acute rheumatic, diphtheritic and scarlatinal heart disease, acquired chronic valvular lesions, and miscellaneous cases.

**CONGENITAL HEART DEFECTS.** Clinically, these cases fall into three groups;: the cyanotic, the potentially cyanotic, and those free from cyanosis. In cyanotic children the stenosis of the pulmonary orifice is compensated imperfectly or not at all by vicarious persistent communications between the pulmonary and general circulations. The non-cyanotic group affords sufficient aeration of the blood via an open intraventricular septum (or foramen ovale) and a patent ductus arteriosus. Thus, as one clinician has paradoxically put it, the greater the number of lesions, the better the condition of the patient. The potentially cyanotic cases, imperfectly compensated by the counter-openings just mentioned, become blue only on exertion or in the course of a disease of the respiratory tract.



The prognosis of congenital heart affections is, broadly speaking, worse according to the degree of cyanosis. I have seen but few cyanotic subjects reach adult life, one of them was forty years of age. They usually succumb in early childhood to some intercurrent disease, with symptoms of asphyxia; most of the survivors show signs of stunted mental and physical development and rarely live beyond puberty, in any event. The non-cyanotic cases do better; I have seen one case recover from a consecutively bilateral pneumonia, followed by empyema; most school children with congenital heart disease belong to this group. Nevertheless, it is remarkable how few of these cases we meet with among adults; we cannot avoid the conclusion that the general prognosis is rather poor for these patients also, though somewhat more favorable than in the cyanotic group. There is less likely to be mental and physical stunting in non-cyanotic children, but I must not fail to mention the relative frequency of tuberculosis and so-called Mongolian idiocy in connection with all grades of congenital cardiac lesions.

Of diagnostic features I may mention the upward extension of the cardiac dulness as a sign of an open ductus arteriosus, and a very loud systolic murmur over the middle of the heart, transmitted upwards as a sign of open communication between the right and left cavities; these symptoms in addition to absence or slowness of cyanosis. A rough systolic murmur in the third left interspace associated with feeble or absent pulmonic sounds is common to nearly all congenital cardiac cases.

Aside from the above types, congenital heart disease is rare and does not call for special discussion here, especially as an exact diagnosis is almost impossible.

**ACUTE HEART DISEASE.** In discussing this group we do best by following the British authors who, under the general name of carditis, regard endo-, myo-, and pericarditis as parts of one clinical entity. As a matter of fact, these patients invariably present endocardial and myocardial lesions at once, sometimes pericarditis in addition, either elicited by physical examination or, as often happens, masked by the general cardiac picture, and therefore not diagnosed.

The prognosis is of course doubtful in all cases, but we may guide ourselves by certain signs. A low systolic blood-pressure,

below 70 or 80 mm., is a danger signal, to be interpreted later on according as it rises or fails to rise in response to treatment. As to the eventual permanent damage to the valves, it is impossible to prognosticate its degree, though a certain residue of injury to at least the mitral valve may be predicted as almost inevitable. Diffuse murmurs are of no great prognostic value, whereas a localized thrill at the apex is almost pathognomonic of an established mitral obstruction.

The question of functional murmurs still remains debatable ground. There is no doubt that soft systolic murmurs, not localized at any orifice nor obliterating the first sound, may disappear without leaving a trace. It is equally true that precisely similar murmurs may gradually localize themselves at the mitral site and become typically valvular in character and significance. It is, therefore, clear that each case must be judged individually, and that prolonged observation is often necessary to determine the diagnosis and prognosis.

The development of accentuation of the second pulmonic sound is evidence of hypertrophy of the right ventricle and consequently of permanent injury to the mitral valve, with obstruction or insufficiency or more commonly both.

If, as sometimes happens, a rough systolic murmur transmitted into the great arteries appears over the aortic site, we know that stenosis of the aortic valve is also developing, with consequent increased gravity of our prognosis.

Myocardial insufficiency as affecting the blood-pressure has already been considered. Arrhythmia is, of course, not to be regarded indifferently, but is far more amenable to treatment than in adults, and does not by itself warrant an unfavorable prognosis, unless it fails to improve and eventually to disappear under proper care.

Pericarditis is a fairly common complication of acute endocarditis, but is frequently overlooked, as its physical signs are often obscure; precordial pain should lead us to suspect its presence, even though the characteristic percussion area and murmur cannot be made out. Pericarditis increases the gravity of the prognosis, chiefly because of the possibility of the development of adhesions; I can recall at least one case, in a boy of thirteen years,

where repeated attacks of pericarditis had caused complete obliteration of the pericardial cavity and death, after the development of an enormous compensating cor bovinum. Imminent danger from the pressure of a large pericardial exudate is not common.

I may mention, in this connection, that acute heart disease of the malignant or septic type, though relatively rare, is by no means unknown in childhood. It cannot be identified positively except by blood-culture, and always involves a grave prognosis.

**ACQUIRED VALVULAR LESIONS.** Whereas, in the adult, a fairly accurate prognosis of valvular lesions is often attainable, this is much less true of children. In the first place, the endocarditis that has caused the valvular damage is far more likely to flare up afresh in early life, and thus increase the damage. Secondly, the compensatory hypertrophy of the myocardium takes some years to become fully developed, and is less likely to prove permanently adequate in childhood than in early adult life. Nevertheless, the prognosis of an uncomplicated stationary mitral lesion, if well compensated, is better than stated in the books; many of these patients attain a good old age and many more become fair risks for life insurance, of course at an advanced premium.

Complication with aortic stenosis or insufficiency renders the ultimate prognosis much worse, as these cases rarely live beyond middle life; the same is true when pericardial adhesions are present. It is true that these lesions may, for a time, be compensated by additional muscular hypertrophy, but this has its limits, and these patients merely use up the reserve that should carry them on to late middle life. Chronic degenerative arterial and myocardial changes may set in before puberty, as shown by one of my patients, who had attacks of true angina pectoris at the age of twelve years.

The movement, lately inaugurated, to make these patients the object of special attention by the school and health authorities, will greatly improve the ultimate prospects of the cardiac pupil, as he will be more or less protected from the evil effects of over-exertion and excessive stair-climbing. On the other hand, it is well to remember that nothing is more predisposing to neuroses and psychoneuroses than the knowledge that one is the victim of a heart affection; I have seen several of such cases in school children, and

the risk of inducing neurasthenia or hypochondriasis is not to be overlooked.

**MISCELLANEOUS GROUPS.** Under this heading mention may be made of certain pathological curiosities. Aortitis and simple aortic insufficiency of the adult type is a great rarity in childhood, not always luetic in origin. As it rapidly leads to great hypertrophy of the heart and impoverishment of the general circulation, the outlook for prolonged life is poor, especially as specific treatment is mostly ineffectual. Considering the frequency of gonococcus infection in children, endocardial complications are very rare indeed. It is well known that, in contrast with the rheumatic type of infection, the gonococcus relatively often attacks the right side of the heart and leads to prolonged disease of the endocardium, which is often of the septic type and not very amenable to treatment. The prognosis is, therefore, very doubtful, both as to imminent danger and with regard to the patient's future.

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## THE DIRECT AND DIFFERENTIAL DIAGNOSIS OF SOME OF THE COMMONER SYPHILIDES

By B. BARKER BEESON, M.D.

Associate in Cutaneous Pathology, Rush Medical College  
Chicago

Nowadays, with so many laboratory facilities at our command, we are at times prone to disregard the value of what can be learned by the sight and touch. Such a tendency should be discouraged, since these were practically the most important means of diagnosis possessed by our forefathers, who were, as we recall, excellent diagnosticians. I trust that no one will mistake this for a slap at laboratory methods, since we must all agree that they are of great value when properly performed and interpreted.

The purpose of this paper will be to emphasize the "naked eye" diagnosis of certain luetic skin lesions, as well as their differentiation from other disorders.

The earliest of the secondary syphilides is the roseola, erythematous syphilide, macular syphiloderm or syphilitic explosion. As

a rule, it is first seen about 45 days after the appearance of the initial lesion. The roseola appears first on the lateral aspects of the chest and flanks, beneath the scapulae and on the anterior surfaces of the forearms. It is composed of macular lesions which are circular or oval in contour and vary from 2 to 10 millimeters as regards diameter, the average being 4 to 6 millimeters. They are of a pale rose color, hence their name. They are non-elevated, do not scale and give rise to no subjective symptoms. The luetic roseola does not reach its maximum development until about a week after its appearance.

Accompanying the roseola is a more or less generalized enlargement of the lymphatic glands. The affected glands are hard, freely movable, painless and sharply circumscribed. As regards size, they range from bean to olive size. The gland nearest the primary lesion is normally the largest and is known as the direct or sentinel gland.

At times the roseola may be mixed with a number of papular elements, and in some cases of lues these may even entirely replace the macular lesions.

The recognition of a characteristic macular syphilide is usually not difficult, but now and then a puzzling case is encountered. Among the diseases which require differentiation are measles, scarlet fever, pityriasis rosea, tinea versicolor, seborrheic dermatitis, ringworm of the glabrous skin, copaiba rash, and the eruption which may rarely follow the ingestion of antipyrine.

Measles is readily diagnosed by its more frequent occurrence in children, onset with marked general symptoms, such as fever, bronchitis and oculo-nasal catarrh. Its eruption is more frankly inflammatory than that of lues and often involves the face, whereas the latter does not. The presence of Koplik's spots on the buccal mucosa would also speak for measles.

Scarlet fever has an acute onset with vomiting, a sharp rise in temperature to 102-105 deg. F. and sore throat. The eruption begins on the neck and spreads rapidly over the trunk, where it is diffuse, and over the limbs. It has a vivid scarlet hue quite different from that of the roseola, which also lacks the accompanying symptoms. The strawberry tongue is also of importance in the diagnosis of scarlatina.

Pityriasis rosea is perhaps mistaken for the specific roseola more often than any other skin disease. This confusion is due to the fact that it is but little known outside of dermatological circles. If its two chief characteristics are kept in mind, the diagnosis is not as a rule fraught with difficulty. They are (1) the presence of medallion-like lesions of which the larger are 2 or 3 centimeters wide and the smaller 8 to 10 millimeters, and (2) scaling. Each medallion consists of a slightly elevated border, 2 or 3 millimeters in width, of a reddish color and exhibiting furfuraceous scaling. The center enclosed within this oval is of a chamois-like color, exhibits slight folding parallel to the lines of cleavage and scales, although there is a tendency to central clearing as the border advances. Their long diameters are placed at right angles to the vertical axis of the body. Pityriasis rosea is not accompanied by enlargement of the lymphatic glands.

The usual case of tinea versicolor, with its *café au lait* or fawn-colored spots, is not difficult to diagnose. Rarely the lesions exhibit a slightly reddish cast. Even in such instances the presence of the branny scaling, the ready removal of the scale *en masse* by the finger nail and the marked chronicity of the disease settles the diagnosis. Seborrheic dermatitis is characterized by greasy scaling, by its frequency on the scalp, where it usually begins, and also on the face, yellowish color, tendency to peripheral spreading, and it is usually accompanied by marked itching.

Ringworm of the glabrous skin possesses circular lesions, which are few in number with an elevated, spreading inflammatory border often studded with tiny vesicles and central clearing, usually located on the exposed portions of the body. Its lesions are larger, exhibit scaling, and usually itch.

The ingestion of *copaiba* is at times followed by a rash which has been mistaken for the luetic roseola. It is composed of plaques of a deeper red color, and is especially marked over the extremities, the lesions being grouped about the extensor surfaces of the knees, elbows, wrists and backs of hands and feet. Marked itching is complained of. The presence of a concomitant gonorrhea, a history of taking *copaiba* and the early disappearance of the eruption after its withdrawal are also valuable points.

Antipyrine may at times give rise to an erythematous eruption

which has been mistaken for the syphilitic explosion. The lesions due to antipyrine are non-elevated, but are larger, due to confluence and prone to group in certain regions such as the face or limbs in contra-distinction to the more disseminated roseola. As regards color, they are of a bright red or vermillion hue. In those who possess an idiosyncrasy to antipyrine, this eruption may appear soon after the initial dose. It often attains its maximum development within a few hours.

The characteristic moth-eaten alopecia of lues and also the thinning of the outer one-half or one-third of each eye brow (sign of the omnibus) are of importance, as are also the presence of mucous patches in the mouth and about the genitals and the generalized glandular enlargement. I have only briefly referred to these, since it is my purpose to devote this article to a description of the skin lesions.

#### PAPULAR SYPHILIDES

This is the most important group of syphilides, not only on account of its numerous varieties, but also because of their frequency and diagnostic value. Papules occur in what is known as the secondary period. They may be concomitant with the roseola, appear soon afterward, or be delayed until several years later. The lenticular papule will be taken as the type for this group, since a resumé of its characteristics when slightly modified will likewise serve for the other luetic papules. An orbicular configuration, a dark red, raw ham, or copper color, and marked infiltration are the characteristic attributes of such a papule. Three to five millimeters is the average diameter of these lesions and they attain an elevation from one-half to one millimeter above the adjacent skin surface.

The circular form of these papules is usually as perfect as though their outlines had been traced with the aid of a compass. This feature is of extreme importance in diagnosis, as is also their marked infiltration, which is best ascertained by palpating a papule between the thumb and index finger, grasping opposite poles. When this is done, it conveys the impression that one is dealing with a tiny neoplasm which has been embedded in the skin. The color, while not so important as the form and infiltration, should not be

forgotten. Papules on the lower limbs present a dark shade of red and may even be violaceous. Usually papules appear in successive crops for one to two weeks.

The papules soon commence to scale and then are called papulo-squamous lesions. Often the scales form a delicate grayish collar around the papule. This has been called the collarette of Bielt. At times the summit of each papule is capped by a slight scale. Instead of remaining dry and merely presenting scales, the papules may become moist and eroded and are then really cutaneous mucous patches or papulo-erosive syphilides. Sometimes in a given case the papules may each be capped by a crust, when they are termed crusted papules. Papulo-pustular syphilides are also seen. As to size, luetic papules may be large or small. The latter is represented by the lichenoid syphilide, in which the papules may be only pinhead-sized. As a rule, the luetic papules are discrete, but occasionally they may be closely packed together.

The papular syphilides can be found on almost any portion of the body, but more especially over the back, the nape of the neck along the hair line, across the forehead, where they form the well-known Corona Veneris, and also about the mouth. They are more generalized than the roseola as a rule. Originally small, they enlarge eccentrically and attain their usual development within a few days. Papular syphilides are prone to recurrence.

The most important conditions requiring a differential diagnosis from the papular syphilides are psoriasis, lichen planus, acne vulgaris, impetigo contagiosa, dry palmar eczema and ecthyma.

In psoriasis the papules tend to merge and form plaques which are most commonly present upon the extensor surfaces of the knees and elbows, over the sacrum, and the scalp. The psoriasiform syphilides are most often lenticular, discrete and tend to favor the palms and soles. The scales of psoriasis are abundant, thick, imbricated and silvery white. In lues they are small, thin, grayish and often form a collarette about the papule.

Upon gentle removal of the scales from a psoriatic lesion, punctate bleeding points are visible. This sign of Auspitz is of value and should not be overlooked, since a similar manoeuvre in lues leads to a more diffuse bleeding. The lesions are much more indurated in lues than in psoriasis.



The lichenoid syphilide or lichen syphiliticus may be confused with lichen planus, but by calling to mind a few of the salient differential points one is soon set aright. The first named is usually present over the back, while lichen planus prefers the flexor surfaces of the wrists and forearms and inner surfaces of the thighs. When lues resembles the disorder now under consideration, its papules, while circular in outline, tend to be acuminate and more closely grouped than in the lenticular variety. They are usually of a raw-ham color, firm to the touch and do not itch. Lichen planus is characterized by papular lesions which are flat-topped, polygonal, have a varnish-like sheen, are of a lilac hue, possess little or no induration and are usually accompanied by severe itching. They tend to be much more confluent than the papules of syphilis and often form veritable mosaics. Upon closer examination, each lichen planus papule is seen to possess a central depression or umbilication. At first lichen planus papules do not present scaling, but even when present it is not so marked as in lichen syphiliticus. When lichen planus present no scaling, or after the scales have been removed, careful examination of some of the larger and older papules will reveal numerous intersecting grayish lines and dots. These have been named the lines of Wickham after the man who first gave them prominence.

Acne vulgaris is another condition which occasionally simulates a papulo-pustular syphiloderm to a marked degree. As to duration, acne is essentially a chronic condition, usually beginning in youth and lasting a number of years. The acneiform syphilides are relatively acute and are much less inflammatory than the lesions of acne vulgaris. They are also much more indurated, smaller, dark red, and usually composed of lesions in the same stage of development. Acne vulgaris is, as a rule, a polymorphous disorder presenting at a given time papules, pustules, comedones and cicatrices. As to location, acne is found on the forehead, over the shoulders, back and supero-anterior portion of the thorax, while its imitator is usually more diffusely distributed.

Occasionally in lues one meets with a case in which the crusting is of such a nature as to simulate impetigo contagiosa. This latter disease is characterized by round, yellowish, honeycomb-like crusts which appear to be stuck on the skin, since they possess no

encircling inflammatory areola. It is most often present in young children and usually affects the scalp, face and hands. Upon removing the crust from an impetiginous lesion, one finds the skin underneath to be slightly eroded, moist and reddish in color. Induration is not present. Impetigo contagiosa is almost always associated with pediculosis capitis. The impetigo-like lesions encountered in lues are frequent on the face and scalp, but are rarely as numerous as those of true impetigo. The lesions in lues tend to coalesce and form plaques which is unusual in impetigo. As is the rule in lues, the crusts are harder and more firmly attached. When they are removed, indurated papular lesions are exposed, which is the most important diagnostic point in favor of impetiginous lues.

One of the most difficult differential diagnoses to make, at times, is that between a palmar syphilide and a dry eczema of that same region. Eczema of the palms is practically always bilateral, while lues is frequently unilateral. In eczema one finds that the eruption is poorly defined, its border is non-elevated and it lacks induration, while on the contrary a palmar syphilide is markedly indurated, and has a well-defined, elevated, often circinate border. Eczema tends to extend from the palm, which it often involves in its entirety, onto the dorsum. Lues does not as a rule affect the entire palm and practically never involves the dorsum by extension from the palmar surface. Individual papules can be found in the active border of a palmar lues.

The crusted papules of lues may be mistaken for ecthyma. This latter disorder is usually associated with some parasitic disorder, such as pediculosis or scabies, and is especially frequent among those who are "run down" and unclean as well. Its most common sites are the legs and the lower portion of the trunk, especially over the buttocks. Ecthyma begins with the formation of vesico-pustules, which are markedly inflammatory and develop in from four to seven days into irregular crust-covered ulcerations. The luetic papule begins as and remains a papule with the characteristics already described. Its development requires from two to three weeks. The ulceration in ecthyma is non-indurated, irregular and inflammatory, while those due to lues are "punched out," indurated, and non-inflammatory.

## NODULAR SYPHILIDES

The above title has been chosen for this class of lesions in preference to that of tubercular syphilides, which could easily lead to confusion with tuberculosis. They differ from papules in being larger, more indurated, more globular as regards form, and they penetrate more deeply into the derma. Furthermore, they are of a dark red or ham color, circular in contour, split-pea to large-bean sized and often later on they are covered by crusts. Nodular syphilides are especially prone to arrange themselves so as to form circles, arcs of circles, crescents, half-moons, and so forth. They occur late in the secondary or early in the tertiary period and are apt to be asymmetrical and localized as a rule to a certain portion of the body, as on the face, especially over the nose, forehead and on the shoulders. At times only six to a dozen nodular lesions may be present, all confined to a small area. Luetic nodules leave brownish macules to mark their sites. These latter lose their pigmentation after a time and become slightly atrophic. As is the case with other specific lesions, those under consideration are characterized by a lack of subjective symptoms. A group of luetic nodules may become serpiginous and advance peripherally with the resultant central scarring. In such a case the active border always contains individual nodules, which usually suffice for diagnostic purposes.

The non-ulcerated nodules of lues must be distinguished from lupus vulgaris. The latter begins in early childhood and progresses much more slowly than does the former disease. By pressing upon a patch of lupus vulgaris with the diascope the characteristic apple jelly nodules are brought to light. They are usually one to four millimeters in diameter, semi-transparent, soft, painful, and of the color already mentioned. These lupus nodules show little or no elevation and may possess or lack scales.

Another disease which must at times be distinguished from nodular lues is acne necrotica or acne varioliformis. It affects by choice the forehead, especially along the hair line, but is also found on the anterior portion of the scalp, about the temples and even over the scapulæ and sternum. This disorder is symmetrically distributed and presents lesions in various stages of development. Acne

necrotica begins as a vesico-pustule which is centrally umbilicated, two to four millimeters in diameter and surrounded by a narrow zone of inflammation. Without opening, this lesion dries and forms a brownish crust, which, when it is detached, exposes a depressed varioliform cicatrix. Thus it can be readily seen that in its lesions and their evolution this disease differs greatly from lues, which is less inflammatory and has lesions which are more indurated, asymmetrical and prone to circinate arrangement.

Lupus erythematodes affects a circular form involving the middle portion of the face, where nodular syphilides are frequent and so may cause confusion. But when one recalls that it does not possess the individual nodules in its advancing border as does syphilis, the diagnosis is made. It is further strengthened by the peculiar central, atrophic stippled-like scarring of lupus erythematodes, which is absent in specific disease.

Nodular syphilides may break down and form ulcers. These ulcers are either very small or the entire lesion may ulcerate en masse just as a gumma would. Such an ulcer is soon covered with a thick, oyster-shell-like, firm, dark-colored crust. These lesions are called rupial syphilides. When the crust is removed, the underlying ulcer is seen to present features which proclaim it to be luetic. It is circular, if several have fused, the outline is polycyclic; it is "punched out," its borders are indurated and adherent. It is deep and the floor is covered by a yellowish core. The whole is seated upon a reddish, indurated base. Such an ulcer would be readily distinguished from one due to tuberculosis if we bear the following facts in mind. Tuberculous ulcers are irregular in contour, with undermined edges, lack induration, are shallow, and purplish in color.

#### GUMMATA

Gummata are late manifestations of lues and occur most frequently from the third to the sixth year after infection, although they have been observed forty to fifty years thereafter. They have four stages—(1) development, (2) softening, (3) ulceration and (4) cicatrization. The lower limbs (42 per cent. of all gummata), arms and head are the common sites for these lesions. Often there is only a single gumma, but at times a number are present in a given area.

A gumma begins as a deep round or oval nodule situated in the sub-cutaneous tissue. Small at first, it enlarges to the size of a hickory nut, olive, or even a small-sized hen's egg, is firm, indolent, distinct from the surrounding tissues, freely movable, and not accompanied by subjective symptoms. By reason of its lack of pain, it is often discovered at this time only by hazard. After a time the gumma approaches the surface of the skin, becomes adherent to the surrounding structures, softens centrally and becomes sensitive to pressure, as well as the seat of spontaneous discomfort. At this time the elevated central portion of the lesion assumes a reddish hue, which soon spreads to the adjacent skin. As the process approaches maturity, this color becomes darker. The skin over the gumma gradually thins, until finally it gives way and the lesion enters on its third stage, that of ulceration.

Gummata are most often observed after they have softened and ulcerated. When this stage is reached, the resulting ulcer, at first small, tends to enlarge. It gives exit to a scanty, viscid, gelatinous, yellowish material resembling mucilage or gum, which has given the lesion its name. The small opening through which the gummatous material escapes is soon enlarged until a circular ulcer varying in diameter from a few millimeters to several centimeters is produced. An ordinary abscess, after it has been opened and its contents evacuated, collapses, but not so with a gumma. Considering the size of gummata, they discharge a comparatively small amount of material at the time of rupture. This is because most of the lesion is composed of its core, a necrotic mass of tissue which has been compared because of its dirty grayish color to cod-fish or may at times resemble gauze which has been left in the ulcer and become saturated with the gummatous secretion. This core is solid, adherent by its base, and is void of sensation. Sometimes when the ulcer has been present for a time the core is not so typical. It may be present only in a rudimentary form as yellowish shreds, which are still attached to the floor of the ulcer. The ulcer is often covered by a yellowish, creamy coating after the core disappears. These three aspects which the floor of such an ulcer may present should be kept in mind. In its early stages the gumma is, of course, more characteristic. After the core has been expelled, the diagnosis must be made by the circular outline and steep,

"punched out" sides. The borders of a gumma are indurated, adherent and dark brown in color. Such an ulcer is 5 to 10 millimeters in depth, even more. The floor is uneven at first, due to the presence of the core. Later on it becomes smoother. Ulcers located on the upper third of the lower leg should be considered gummata until proven to be otherwise. Finally, either spontaneously or as a result of treatment, the ulcer assumes a more healthy appearance and healing begins. The lesion becomes shallower and its diameter steadily decreases. At length cicatrization is complete and the typical scar remains to mark its site. This is of a reddish violet color at first, becoming paler as time goes on. Its edges, especially in gummata on the legs, remain pigmented for a long time. Finally a colorless, parchment-like scar remains. It is somewhat depressed, thin, smooth and supple. The average duration of a gumma is 3 to 4 months.

Now and then a lipoma, fibroma or sebaceous cyst has been mistaken for a gumma in the process of development. All the above are very chronic and do not evolve as the gummatous lesions do. When any of them become infected and suppurate, the resulting lesion does not present the characteristics of a gummatous ulcer.

Sporotrichosis can readily cause confusion if one is not on the quiver. Its lesions are more numerous than gummata as a rule. They do not show such a marked predilection for the lower extremities and their development is more rapid. In 6 to 8 weeks they break down and collapse more or less completely after their contents have been discharged. They possess no core. The ulcerations in sporotrichosis are polymorphous, some resemble those of lues, others may simulate tuberculous lesions, while still others are ecthymatiform. They are smaller than gummatous ulcers and are often more like fistulæ. Their borders are irregular, undermined and violaceous. Autoinoculation is frequent.

An ordinary abscess develops much more rapidly than a gumma. It is accompanied by marked pain, which may be of a lancinating character. Fever and enlarged lymph glands are also present. After such a lesion has been incised, healing soon takes place.

Tuberculous abscesses are almost always secondary to osseous or glandular disease. They are frequent about the neck (scrofuloderma). They develop very slowly and possess no core. Such an

ulcer would be fistulous, violaceous, atonic, with irregular borders and soft undermined edges.

Carcinoma is characterized by an early adherence to the skin, as well as glandular enlargement. Gumma does not cause enlargement of the lymphatic glands. The former becomes adherent to the skin at any earlier date than the gumma. Carcinomatous ulcers are irregular with everted edges of a wood-like hardness. The characteristic "cancer" pearls are often present in the border. The floor of an ulcerated malignant growth has a fetid secretion and bleeds upon the slightest touch.

One of the most important, as well as difficult, conditions to distinguish from gumma is what is known as varicose ulcer. When confronted with such a lesion, one should always think of lues as the possible causative factor. An ulcer found upon a leg showing varicose veins is not necessarily a simple varicose ulcer. It has been stated that at least 20 per cent. of such ulcers are really luetic. Only by close attention to the details of these lesions can a diagnosis be arrived at. First, as to location, varicose ulcers are most commonly present in the lower half of the leg, especially about the internal malleoli. Gummata are found in the upper half, often close to the patella.

Varicose ulcer is most often unique, while several gummata are usually present. A large ulceration surrounded by satellites is almost always luetic. As to configuration, the simple ulcer does not present the characteristic circular form which at once causes the careful observer to think of syphilis. It is irregular and even when round does not look so mathematically exact as does the gumma. Specific lesions are also reniform, semilunar and horse-shoe shaped. The floor of a varicose ulcer is reddish yellow, grayish or violaceous. It may be covered by a purulent secretion, but does not at any time possess the core which has already been described. This is of extreme importance in diagnosis. Thick elevated borders with sloping sides are present in varicose ulcers in contradistinction to the thinner, "punched out" margins of a gummatous ulcer.

Gummata possess an indurated surrounding zone, but it is much less in extent than that found in association with varicose ulcers. These latter are surrounded by an edematous pigmented zone which may involve the greater part of a limb. Lastly, the crusts of luetic

gummata are thick, stratified and dark brown in color and very adherent, varicose ulcers present little or no crusting.

I have not undertaken to describe all the cutaneous affections which may simulate the protean lesions of lues, but have merely touched upon certain of the more common ones. The presence of concomitant luetic lesions should always be sought for when making a diagnosis of any skin disease. When present they are of prime importance. It may seem as though much which is common property has been discussed. While this is undoubtedly true, I believe that we cannot over-emphasize the importance of lues nor can the physician become too skilful in its detection.

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## EXOPHTHALMIC GOITRE

### REMARKS ON THE SYMPTOMATOLOGY, PROGNOSIS, AND THE NONSURGICAL TREATMENT

By ISRAEL BRAM

Philadelphia

#### INTRODUCTION

Exophthalmic goitre, hyperplastic goitre, Graves disease, Parry's disease or Basedow's disease, is a chronic, rarely an acute affection, thought by most observers to be caused by overaction of the thyroid gland. Its chief manifestations are the classical cardinal symptoms—tachycardia, tremor, exophthalmos and enlarged thyroid.

There is no morbid state of the body which has enlisted more painstaking attention of biologists, pathologists, physiologists, internists, surgeons, and even of the philosophically inclined elements of the laity, than exophthalmic goitre. There is no disease more mysterious in manifestations, more fascinating in study, more provocative toward diversity of etiological theories, and more refractive to conclusive analysis than is exophthalmic goitre. Is it any wonder that men wrangle to-day as much as ever over the various aspects of hyperthyroidism, and is it surprising that volumes upon volumes of literature have been written upon so evasive a subject?

But though we understand little concerning the formative mechan-



ism of the syndrome, we already know through careful observation much of the etiology of the disease, especially of the predisposing factors. We may also pride ourselves on a knowledge of a few very ingenious theories to explain the exciting factors which bring on the syndrome, though these theories are mutually antagonistic and may all be proved wrong in the near future by some enterprising young laboratory enthusiast. The sufferer from the disease, however, is not much interested in the etiology or pathology of the condition; what concerns this individual is relief or cure. And here is where the patient may truly gain comfort, for relief, even cure, is almost positive in the vast majority of cases if treatment is instituted early enough, i. e., before the organs involved in the syndrome are markedly damaged.

#### RELATION OF SIMPLE TO HYPERPLASTIC GOITRE

Variations in the gland size and even function without appreciable symptoms are so common that it is often impossible to sharply differentiate between a normal and an abnormal thyroid. There is reason to believe that the gland has a storage function which, if carried to excess, leads to local areas of stasis through pressure atrophy of the producing cells. It is such glands that are classified under simple or nontoxic or atoxic goitre. Colloid substance is thought to be that substance which serves to hold the gland contents. In exophthalmic goitre, however, there is always a true hypertrophy and hyperplasia, that is, an increase both in cells and in activity of the gland.

Cases of simple goitre under excitement or other unknown causes begin to pulsate presenting vaguely some suggestions of Graves disease, which may disappear with proper rest and equanimity of body and mind, the case soon assuming the picture of simple goitre. These are the "borderline" cases, and the frequent recurrence of the syndrome, be it ever so mild, may soon terminate in permanent Graves disease. In some instances a simple goitre may gradually become exophthalmic goitre without reverting back to the simple type, and without known cause.

Generally speaking, though the cause of simple goitre is not the cause of Graves disease, any abnormality of the thyroid gland may instigate a hyperplastic process with consequent symptoms of hyper-

thyroidism on the one hand, or a hypoplastic process, with resulting symptoms of hypothyroidism, on the other.

#### SYMPTOMS AND PHYSICAL SIGNS

##### (a) General

The rapid, acute, "abortive" type of Graves disease is fortunately not often encountered. A case of J. H. Lloyd's, quoted by J. M. Anders, proved fatal after an illness of three days. The course is rather malignant; there is marked, rapid loss of weight. The patient may lose one-half the body weight within a few weeks. The most constant symptom is the tachycardia, usually associated with distressing palpitation which may lead to arrhythmia. There is high blood pressure, occasionally fever and severe gastro-intestinal symptoms with incessant vomiting and purging. The exophthalmos is marked, and at times there are cerebral symptoms leading to one of the various types of mental aberration.

A subject of the chronic or usual type of the disease may appear in the doctor's office complaining merely of muscular weakness of a few weeks' duration, with progressive loss of weight; or the chief complaints may be occasional palpitation, nervousness, and insomnia; or in some instances the dominant subjective feature may be frequent diurnal and nocturnal micturition which causes the subject to suspect diabetes or Bright's disease. Occasionally marked sweating, especially at night, will become so regular and exhausting, that the patient will fear the existence of tuberculosis and will consult the physician with this diagnosis in view. Indeed, the manifestations of this disease may vary so greatly from the classical syndrome that only too often the patient, and even the busy general practitioner, will diagnose the case under such headings as "nervousness," neurasthenia, "run down system," nervous dyspepsia, anemia, pulmonary tuberculosis, and even diabetes mellitus. There is no disease in the domain of medicine more likely to be confused with some other condition than hyperthyroidism, and in due justice to the diagnostician, we must remark that an erroneous diagnosis is not without cause, as there is no condition in medicine more capable of successfully mimicking so many totally different diseases. The peculiarly persistent increase in temperature in marked hyperthyroidism tends further to lead the diagnostician from the correct diagnosis toward

the consideration of a more common morbid process. Many of these patients experience a definite rise in temperature to 99.5 or even 100 toward evening, followed by drenching sweats.

The marked bodily wasting is not only an early symptom of hyperthyroidism, but is a conspicuous accompaniment of the other manifestations during the entire course of the disease. This marked wasting is probably due to the well known oxidizing quality of thyroid secretion, first destroying fatty, then albuminous tissue, and is probably an action analogous to that claimed for it in the destruction of albuminous toxins of the intestines.

The acceleration of metabolism may be satisfactorily demonstrated by the administration to anyone of the extract of thyroid gland, when the reduction in weight in the course of a few weeks will equal that of any case of Graves disease. With the great loss of weight there is considerable anemia of the chlorotic type, which, because of the characteristic flush of the skin (due to slight rise in temperature and vasomotor instability) is not discernible on inspection.

#### (b) The thyroid gland

A case of Graves disease does not necessarily mean an associated thyroid enlargement. Indeed, many cases of marked hyperthyroidism never present any appreciable thyroid hypertrophy. However, it must be remarked that in such instances there are thyroids which instead of symmetrically increasing in size, grow "inward," that is, there is hypertrophy of that portion of the gland in contact with the trachea. In such instances, patients will not present visible thyroid enlargement, and yet will complain of choking sensations because of pressure, and rarely of a dry hacking cough. Ordinarily, thyroid enlargement, varying from a slight fullness to an appreciable goitre, will precede, accompany, or follow exophthalmos and tachycardia.

The enlargement is moderate, general or partial, the size presenting variations depending chiefly upon the circulatory disturbances within the thyroid gland. Usually there is a smooth, rounded, diffuse enlargement, rarely attaining the size commonly seen in subjects of simple atoxic goitre. Close inspection may in severe cases present a visible systolic throbbing, and the enlarged superficial veins may be seen coursing about under the skin.

On palpation tenderness is usually elicited (Kocher). The degree

of symmetry is determined in confirmation of inspection. In my experience there is usually a greater enlargement of one lobe of the thyroid as compared with the other, and somewhat out of proportion with the enlargement of the isthmus. The mass is usually soft and yielding, without nodules, but occasionally marked resistance is offered to the palpating fingers. This is due to fibroid changes within the gland, but in aged subjects the possibility of malignant degeneration must always be borne in mind. In many well developed cases of Graves disease a distinct systolic thrill simulating that observed over an aneurism is felt. On auscultation a systolic murmur is heard in most cases, especially where the thrill is felt. Occasionally the murmur is somewhat similar to the see-saw murmur heard in a double mitral lesion, excepting that here the sounds are somewhat harsher and less musical in quality.

### (c) The eyes

The important eye manifestation in this disease is exophthalmos, which occurs in approximately 90% of cases, and is probably due to an increased blood and lymph supply in the orbit, plus an increased amount of orbital adipose tissue. It may occur early or late in the syndrome of symptoms; a slight bulging of the eye-balls may be the only evidence for a long time that there is something wrong with the patient, and this may prompt the subject to seek medical assistance, at which time the presence of other evidences of hyperthyroidism may clinch the diagnosis. The exophthalmos may be unilateral at first, or very rarely, permanently so. In cases not so severe, the bulging may be within physiological limits (Heerfordt). Excessive epiphora may precede for a time the appearance of exophthalmos (Berger). Exophthalmos may vary in degree at different times in the same patient, and the degree of the exophthalmos bears in relation to the size of the goitre or the severity of the other symptoms (Dock). The approach of the menstrual period may accentuate this symptom, the exophthalmos remaining unusually marked until the menses have passed. Physical, and especially mental excitation may increase the protrusion of the eyes until they seem ready to "pop out." Indeed, in rare cases there is no discernible cause for a rather malignant progress of this exophthalmos in which though the progress of other evidences of the disease has apparently been

checked, the eyes continue to bulge more and more forward, leading to very sad consequences. I have seen a case in which a double enucleation became necessary to save the life of the patient because of a virulent infection of the eye-ball resulting from ulceration through congestion (passive) and exposure. In a recent case of mine which completely recovered, the patient was obliged to place small pads of lint over her eyes at bed time, for protection from exposure. Through patient, untiring efforts, by means of hygienic, dietetic, medicinal, and electrotherapeutic measures, the amelioration of her condition brought about a complete recession of the eye-balls to normal within six months.

Ulceration of the cornea, as intimated, is not an uncommon occurrence. The function of the eye-lids as protectors of the delicate ocular conjunctiva being diminished, the lids being unable to close over the protruding organ, the eye-ball is exposed to the air day and night; even winking is for that reason less frequent and less complete. As a result of this exposure, chronic conjunctivitis and corneal ulceration may occur, leading frequently to partial or complete blindness, and if infection becomes marked, to panophthalmitis and an urgent necessity for enucleation to save the patient's life.

Ophthalmoscopic changes are not marked or typical. There may be a dilatation of the arterial vessels, their calibre becoming equal to that of the veins. Arterial pulsation is frequently discernible.—(Beckner.)

Difficulty in everting the upper lids is an almost constant sign.—(Goffre.)

Pigmentation of the upper lids is characteristic of the disease.—(Jellinek and Rosin.)

Stellwag's sign (staring eye) consists of imperfect power of winking or diminished frequency of the act. There may be a number of rapid winks succeeded by a long pause in which there is no movement of the lids. Each wink is incomplete; the margins of the lids do not come together as in the normal eye. This absence of normal involuntary winking may be an early sign, the patient not winking for minutes at a time, and then incompletely.

Dalrymple's sign consists of retraction of the upper lid so that there is an undue separation between the two lids. The resulting

widening of the palpebral fissure produces the peculiar stare which is present in Graves disease, somewhat similar to the effect produced by cocaine.

Von Graefe's sign is important in the early recognition of the disease. In a normal eye, when the globe is directed downward the upper lid moves in perfect accord with it. In subjects of Graves disease the upper lid follows tardily the downward movement of the eye-ball, or in some cases does not move at all. This symptom is not constant, but is almost always a precursor of exophthalmos, and persists after the subsidence of protrusion of the eye-balls.

Moebius' sign consists in a diminution or absence of convergence. To test the convergence near point approach a finger or pencil to the nearest point upon which the eyes can converge, which normally should be situated at no greater distance than three and one-half inches from the eyes or about one and one-half inches from the nose. If outward deviation of an eye occurs before this point is reached, convergence is deficient. Paresis of the ocular muscles is more characteristic than mere insufficiency of convergence (Heerfordt).

Kocher's sign consists in a slight momentary retraction of the upper eyelids on gazing at some object if the latter is moved up and down.

Tremor of the eyeballs is an occasional marked sign. An almost constant sign is the presence, on closing the eye, of a rim of white above and below the cornea.

Curschman describes a dissociated reaction of the pupils (epinephrin mydriasis) which he has observed in a considerable number of cases.

Subjective feeling of pressure behind the eyes (A. Kocher) and abnormal dryness of the eyes (Preble) are also worthy of note.

#### (d) Circulatory system

A rapid heart is a suspicious early symptom, often preceding for a long time the other cardinal features. The beats become unusually excitable, violent, and irregular on the slightest provocation, often reaching as high as 160, or even 200 per minute. Palpitation may become rather troublesome, and may be the first and only symptom complained of. Dyspnea, resulting from this cardiac excitation

frequently reaches the point when the patient, extremely "short in breath" on going up stairs, imagines that there is some pulmonary disease.

Diminished chest expansion, and as the disease progresses, a chest contracted in the antero-postero diameter, are highly characteristic of Graves disease. The dyspnea of the diminished expansion, plus the picture of the contracted chest are frequently the cause of a tentative anagnosis of pulmonary tuberculosis, and may actually lead in some cases to a complicating phthisis. Inspection presents at first a feeble apical impulse, not displaced. Later, as hypertrophy and dilatation occur from cardiac overactivity, the cardiac area becomes much extended. I have seen cases in which the greatly overacted, enlarged heart has caused the head to nod with each cardiac cycle.

The large vessels, especially the carotids and the abdominal aorta beat violently, resembling in this respect the picture presented by aortic regurgitation. The capillaries and the veins of the hands and neck may also beat visibly.

The location and character of the apex beat, the throbbing arteries, and the diminished respiratory expansion are corroborated by the palpating fingers. A distinct thrill may often be felt over the cardiac area, especially if dilatation has advanced to the point of valvular insufficiency.

Percussion reveals a gradually increasing extension of the cardiac area as the disease progresses. A marked hypertrophic dilatation may cause the cardiac area to extend to the axillary space. Careful percussion over the upper part of the sternum may reveal the presence of an enlarged thymus gland.

Auscultation at first reveals nothing of importance excepting a more forcible, excellerated apex beat and possibly a hemic systolic murmur. Later the exhaustion of the myocardium gives rise to a beat the quality of which is weaker than normal. Progressing to relative mitral insufficiency, murmurs, at first apical, systolic in time and transmitted into the left axillary space, then an apical presystolic, finally a tricuspid and an aortic murmur are apt to develop, leading to general anasarca and occasionally to death from heart failure.

A systolic bruit over the great blood vessels, especially the sub-

clavian, carotid and abdominal arteries is quite common in this disease, especially during its most active stage.

The radial pulse is small, rapid, and of poor volume. In advanced cases with cardiac embarrassment arrhythmia may occur.

The blood pressure is at first high, mounting up to 180 m.m. or more in early cases. With the weakening of the myocardium and occurrence of circulatory relaxation the pressure is lowered, reaching 130 or 120 m.m., and as this process continues to cardiac insufficiency, the systolic blood pressure may come down to 100 or even 90 m.m., associated with considerable cyanosis and dyspnea. The blood is usually profoundly influenced through hyperthyroidism. The intoxication, the poor respiratory functions, the poor nutrition all conspire to lead to blood impoverishment which on inspection may not be evident. The increased temperature of the patient and the vasomotor instability may give rise to rosy cheeks, masking a severe chlorosis. A blood analysis not only reveals a marked diminution of hemaglobin with a moderate reduction in red blood corpuscles, but there is usually a definite leucopenia, i. e., a reduction of neutrophiles and a relative increase in lymphocytes.

Hemorrhages from the nose, stomach, lungs and bowels may occur, often to a dangerous degree.

#### (e) Nervous system

The most marked, characteristic and constant nervous manifestation of hyperthyroidism is the tremor of the outstretched fingers and toes. This tremor is fine, involuntary, and about eight to twelve to the second. So constant is this symptom that it has been my rule to request patients who present a slight degree of heart rapidity or history of loss of weight, to extend their arms and stretch out their fingers; in this way I have discovered many a case of incipient hyperthyroidism which has gone the rounds of physicians and clinics for neurasthenia and other erroneous diagnosis.

Of course, a tremor of this sort unaccompanied by at least one other characteristic symptom of hyperthyroidism especially an undue fulness of the neck, staring of the eyes, or an afebrile tachycardia, should not necessarily mean Graves disease, and the course of the patient's condition should be carefully observed. Occasionally, especially in early cases, the tremor may be limited to one or two fin-



gers of one hand, and in some instances I have seen a slight fine tremor of one or two fingers followed within from 30 to 60 seconds by the tremor of several other fingers. The patient must be kept in this posture for fully a minute.

There comes over the patient in many instances a gradual strangeness of disposition—a change of habits and taste, which leads to the conclusion by the members of the family that “there is something wrong” or that the subject is “terribly nervous.”

The patient is easily excited; the slightest noise, a sudden call, a slight peal of thunder, and the exophthalmos is markedly accentuated; the heart thumps away at a terrific rate, and the features become distressed and anxious. These patients are easily aroused to the extreme of almost any emotion; fright, anxiety, terror, suspicion, anger, may alternate with surprising rapidity and upon the most trivial provocation.

Neurasthenia, or rather the manifestations characterizing neurasthenia, are quite prevalent among subjects of hyperthyroidism. Headache and vertigo may for a long time precede the other nervous manifestations. These patients, especially young adults, are easily fatigued.

The condition may in some instances merge into a state of mania or melancholia, and rarely hallucinations, persecutory delusions and delirium. Insomnia may be caused by the marked throbbing of the heart and blood vessels, by excessive sweating, by mere “nervousness,” or as has often occurred in my experience, by the incessant demand for micturition.

#### (f) The skin

Edema, circumscribed or general as a sequence to cardiac weakness, is commonly present in advanced cases presenting leaking cardiac valves.

Marked diminution in cutaneous resistance to electrical currents is an almost constant symptom (Charcot).

The temperature of the skin in subjects of hyperthyroidism is at times elevated. It is thinned in texture, more sensitive, often dermatographic. Peripheral stimuli are more rapidly conducted, more acutely and intensely translated, and the responses are exaggerated.

Hyperidrosis or excessive sweating seems to account for the increased electric conductivity of the skin (Vigouroux).

Indicating loss of accipito-frontalis control, the forehead is smoother than in health with failure of the forehead to wrinkle on looking up (Joffroy).

Vasomotor instability often resulting in prominent erythematous blotching of the neck and chest, is a conspicuous sign.

Pigmentation, often so marked as to resemble Addison's disease may occur, and occasionally one may see instances of pruritis, urticaria and scleroderma.

Of special diagnostic importance is the fine pin point petechiae, most often occurring over the front and upper portion of the chest.

#### (g) Gastro-intestinal system

Anorexia, though common, is not a constant symptom. Some patients possess a normal appetite, and occasionally a sufferer from hyperthyroidism will have the ravenous appetite characteristic of diabetes mellitus, in spite of which there is gradual loss of weight.

Nausea and vomiting simulating nervous dyspepsia are quite commonly seen and may be attributed to the nervous instability. During an attack of acute hyperthyroidism, and in the advanced stage of the usual chronic type of the disease the vomiting, partially caused by gastric achylia, may become so severe as to precipitate a fatal termination through exhaustion.

Though in most cases of hyperthyroidism the bowels are normal in action or there is slight constipation, in a small percentage of instances there is diarrhea, and in advanced stages (possibly due in part to passive congestion of the intestinal mucosa), the purging may become alarming and cause more concern than any of the other manifestations of the disease.

Fatty stools and undigested meat fibers in the feces are frequently present, resembling the findings in diabetes mellitus. This is probably due to the associated glycosuria, and suggests a degree of pancreatic and possibly biliary disturbance.

#### (h) Urinary system

The accelerated metabolism characterizing hyperthyroidism gives rise to an increase in the output of the urea-nitrogen, total nitrogen, uric acid and the phosphates of the urine. Albuminuria (usually transient) is often present, and is due to a co-existing nephritis, an irritation of the kidneys by the oversupply of thyroid substance

in the blood, the passive congestion consequent upon cardiac incompetency, or to a combination of these causes. It rarely assumes alarming proportions, and is occasionally accompanied by hyaline and granular casts.

Polyuria is a frequent symptom. There is not only in these cases a greater total output of urine, often approximating the quantities observed in diabetes mellitus, but there is also an undue, quite troublesome frequency of micturition. It seems that the bladder also participates in the general nervous excitation and is incapable of retaining more than small quantities at a time. In a patient of mine, it was necessary to arise from ten to fifteen times every night to micturate. No wonder he suffered with insomnia!

Glycosuria. Alimentary glycosuria is rather common in the presence of a hyperactive thyroid gland, occurring in approximately 85 per cent. of my cases. This is probably due to a lowering of carbohydrate tolerance, and the more severe the case, the greater this intolerance and the higher the glycosuria. Indeed, so marked does this become, that it is often necessary to greatly reduce or even to withdraw for a time all starchy foods, and to temporarily treat the case as one of diabetes. The artificial production of hyperthyroidism through the administration of thyroid extract is also productive of glycosuria, which fact leads to the conclusion that it is not a primary change in the Islands of Langerhans but the presence of an undue quantity of thyroid substance in the blood, that is responsible for the presence of sugar in the urine.

#### DIFFERENTIAL DIAGNOSIS

The cardinal symptoms are enlarged thyroid, exophthalmos, tremor of the out-stretched hands and tachycardia. In the presence of these four, the diagnosis is simple, and the case is merely one of therapeusis. But because of the frequent absence of certain cardinal symptoms, the diagnosis of Graves disease is often fraught with difficulties. In many cases the patient is questioned and examined with a view to the discovery of some other more common morbid condition, and hyperthyroidism is not thought of. Though exophthalmos and enlarged thyroid put the diagnostician immediately on his guard, these symptoms in early cases are either entirely absent or so slight as to be entirely overlooked. On the other

hand, exophthalmos is occasionally the only feature present for a long time. But cardiovascular symptoms occur early, and a subject of hyperthyroidism is rarely if ever free from continuous or spasmodic occurrence of palpitation, tachycardia or arrhythmia.

The unaccountable persistent tachycardia, the palpitation associated with the drenching night sweats unassociated with the physical signs of tuberculosis are always highly significant. Persistently increased frequency of urination and polyuria, especially in a young woman of highly neurotic temperament who complains of paroxysmal palpitation and intractable diarrhoea must be considered at least suspicious of Graves disease, as these symptoms are frequently forerunners of a typical development of hyperthyroidism.

What morbid conditions must be excluded in the differential diagnosis of Graves disease? Let us begin with *simple goitre*:

*Simple or Nontoxic goitre*

(a) Tendency to distinct circumscription, often beginning as an egg shaped tumor over the isthmus; tendency to progressive growth, often assuming markedly noticeable proportions.

(b) Comparatively rapid growth.

(c) Pressure symptoms common.

(d) Pulsation, thrill and bruit rare.

(e) Frequently a history of goitrous geographical location or numerous pregnancies, not dependent upon shock or neurotic tendencies.

(f) No cardiovascular, nervous, ocular, gastrointestinal, cutaneous or urinary manifestations.

(g) No change in thymus.

*Exophthalmic goitre*

Enlargement is diffuse, smooth, round, often a mere thyroid fullness, rarely assuming a size perceptible at a distance.

Comparatively slow growth.

Pressure symptoms rare.

Pulsation, thrill and bruit characteristic.

History of nervous shock, fright or a neurotic tendency.

Characteristic cardiovascular nervous, ocular, gastrointestinal, cutaneous and urinary changes.

Thymus enlargement.

- |   |   |
|---|---|
| (h) Usually no weakness or emaciation.                                  | Marked weakness and emaciation.                                 |
| (i) Often improved by administration of thyroid extract or the iodides. | Symptoms markedly aggravated by thyroid extract or the iodides. |

*Pulmonary tuberculosis.* The rise in temperature, persistent sweating, diminished respiratory expansion, weakness, and loss of weight frequently lead doctor and patient to suspect phthisis. The following tabulation will assist in differentiating, though occasionally the two conditions exist in the same patient.

*Pulmonary Tuberculosis*

- (a) No thyroid fullness, tremor, or exophthalmos.
- (b) Usually a history of exposure to the disease directly or indirectly, or a tuberculous family history.
- (c) Cough, expectoration, vague chest pains and often Koch's bacillus in the sputum. Tuberculin reaction. Characteristic physical signs over the lungs.
- (d) X-ray examination reveals pulmonary lesions.

*Exophthalmic Goitre*

- Thyroid fullness, tremor and exophthalmos.
- Not so. Frequently a history of nervous shock or neuropathic tendency.
- Not so.
- X-ray examination may reveal enlarged thymus and dilated heart.

*Diabetes Mellitus.* The glycosuria, polyuria, fatty stools and rapid wasting of a subject of Graves disease may closely resemble diabetes mellitus, and unless the patient is carefully observed for several weeks the proper diagnosis will never be made. Polyuria may for months be a forerunner of exophthalmic goitre. Glycosuria may be temporary or slight, accompanying the usual Graves syndrome, or the disease may assume the typical picture of diabetes mellitus. Moreover, diabetes may co-exist in a subject of exophthalmic goitre, gain the upper hand, and in rare instances a patient may die of diabetic coma. The absence in true diabetes mellitus of thyroid

fullness, tachycardia, exophthalmos, tremor, moist clammy skin and rise of temperature, and, on the other hand, the absence in hyperthyroidism of *persistent* large quantities of sugar in the urine, very great output of urine, great hunger and thirst, itching, dry skin, and visual disturbances, will assist in differentiating the two conditions.

*Hysteria* and *neurasthenia* are often with difficulty differentiated from early hyperthyroidism because of the manifold nervous symptoms occurring in the latter. The tremor, restlessness, mental irritability, insomnia, polyuria, nausea, vomiting, diarrhoea, loss of weight, and numerous minor details may resemble a nervous disease of functional type. "Nervousness" is commonly the diagnosis made when a very early case is first seen. Alertness of observation is highly necessary in order that no time be lost in instituting appropriate remedial measures.

*Anemia*, primary or secondary, is easily differentiated from Graves disease by the presence in the latter of the characteristic symptoms. As mentioned elsewhere, there is a degree of chlorosis in the typical case of hyperthyroidism, though this may not be evident on inspection.

*Septic endocarditis* is sometimes thought of when the symptoms of hyperthyroidism are acute, and in the presence of a frenzy or delirium, so to speak, of cardiac function, marked prostration, sweating, feverishness and great weakness, it becomes a rather difficult matter to rule out septic endocarditis. A careful history will usually reveal in the latter case the presence of a preexisting infection, such as acute articular rheumatism, lobar pneumonia, scarlet fever, typhoid, erysipelas, etc. There is marked pain over the abdomen and elsewhere, evidences of embolic processes, scanty febrile urine, frequent chills, leucocytosis, the frequent presence of septic organisms in the blood, early cardiac murmurs, rapid onset of the typhoid state, and the absence of thyroid fullness and tenderness and exophthalmos.

*Paroxysmal tachycardia* occurs at intervals, the pulse is usually more rapid than in Graves disease (usually about 200 or more per minute) there is usually a sense of great cardiac discomfort and a sense of impending danger, but heart rate becomes suddenly

normal within from a few hours to several days, and there is the absence of the typical symptoms of hyperthyroidism.

*Addison's disease* must in rare instances be differentiated from Graves disease when marked pigmentation occurs in the latter. James M. Anders ("Practice of Medicine" 12th ed.) recently saw such a case in a physician.

*Paralysis agitans* is sometimes thought of, because of the tremor. The tremor in this disease is much slower than that of Graves disease and is characterized as "pill rolling," ceasing for a few moments after voluntary movement is begun and during sleep. Aside from the tremor, the diagnostic features of the two conditions are so distinct that a detailed outline is unnecessary.

#### COURSE AND PROGNOSIS

The course of exophthalmic goitre is difficult to outline with definiteness and precision, because of the variability of the types of the disease. In general, it may be said that the seriousness of the case depends upon the severity and number of the symptoms, the duration of the affection, and the presence or absence of complications, and promptness with which adequate treatment is instituted. The sex must also be considered; a more aggravated form of the disease is frequently seen in men.

The acute cases (fortunately rarely seen) may die in from a few days to a month or two. Some acute cases recover rapidly or merge into the chronic type; on the other hand, a chronic case will occasionally assume a malignant course and become acute with rapid fatal termination.

A certain percentage of cases (10 per cent.—Dock) die as a result of intercurrent diseases, i. e., heart failure, nephritis (of passive congestion), syncope, or from the exhaustion of emaciation, gastrointestinal disturbances, hyperidrosis, insomnia, or from a complicating pneumonia or phthisis.

The chronic form of Graves disease usually continues on for years. An untreated case may present a gradual subsidence of symptoms. This occurrence is rare, and it is, due, I believe, to degenerative changes within the hyperfunctionating thyroid gland, resulting in a restoration to normal of thyroid secretion formation. This may explain the occasional spontaneous recovery. In some instances,

this degenerative process may continue on to past the point of physiological equilibrium, and the continued incapacitation of thyroid parenchyma may lead to hyposecretion and evidences of myxedema. This is the probable mechanism of the course in those instances of hyperthyroidism which in course of time evince symptoms of hypothyroidism.

#### COMMENTS ON NONSURGICAL TREATMENT

In the N. Y. Med. Jour. of November 27, 1915, and April 28, 1917, I stated that Graves disease should be considered strictly in the domain of medicine, and that the surgeon steps in only in the presence of malignant changes in the thyroid, or where this organ becomes so large as to cause dangerous pressure symptoms.

It is readily conceded that malignant changes in the thyroid gland, as cancer anywhere in the body, is a non-medical status. But cancer of the hyperfunctionating thyroid is a rare condition; I have seen but one case in an old woman who was permitted to drift on for many years without attention of any kind.

Severe pressure symptoms rarely occur in Graves disease, and when pressure over the trachia is experienced the timely institution of the proper nonsurgical measures has always yielded relief in the course of a few weeks in my experience. What are my concrete objections to surgical measures in the treatment of exophthalmic goitre?

1. Surgery does not *cure* Graves disease. This is attested by such men as Berry ("Diseases of the Thyroid Gland"), and implied in the statements of Musser (Am. Jour. Med. Sci., June, 1912), Hall, White and Mackenzie. "The sympathetic operation may slightly diminish exophthalmos, but may be followed by very serious results, such as inflammation of the eye or even blindness. Regarding the ligation of the thyroid vessels, it still seems doubtful whether this procedure is followed by cure sufficiently often to justify its performance."—Berry.

2. The contention that the operative mortality is less than five per cent. does not mean recovery from Graves disease, as any fair-minded surgeon will admit. Surgical improvement does not stand the test of time; in my inquiries of a dozen eminent surgeons concerning complete surgical cures of Graves disease, I have not been



able to secure satisfactory information about a single instance of surgical *cure* of Graves disease. In my own record of thirty-two cured cases, three were previously operated on with excellent surgical results, but with failure of cure, the symptoms having recurred with a vehemence that necessitated the closest observation for several weeks. Partial thyroidectomy leaves part of the thyroid, from which sooner or later the goitre redevelops with the reappearance of the syndrome.

3. The frequent presence of sugar in the urine is a contraindication to surgical procedure.

4. The administration of general anesthetic or even local anesthesia in a patient whose vital functions, especially the nervous system, are already demoralized tends to aggravate matters and is therapeutically inconsistent. The surgical shock in the presence of poor circulatory and renal functions adds "insult to injury."

5. The frequently occurring post operative myxedema as a result of thyroid enucleation is worse than Graves disease. Kocher states that the average life of a case of myxedema is seven years.

6. Apparent surgical cures are due to post operative nonsurgical treatment. All reliable surgeons routinely refer their cases back to the family doctor for post operative treatment, and the success or failure of the outcome depends upon the degree of intelligence with which these post-operative dietetic, hygienic and medicinal measures are adopted.

7. The occasional removal of the parathyroids during a thyroidectomy with resulting tetany, is by no means a rare occurrence.

8. Injury to the recurrent laryngeal nerve directly through the operation, or subsequently by pressure of scar tissue, is not uncommon, leading to paralysis of the vocal cords.

9. The surgical mortality rate is higher than the stated five per cent., while the nonsurgical mortality if treatment be not too greatly postponed, is practicably nil. In my series of thirty-two cured cases of Graves disease extending over a period of six years there has not been a single recurrence, and as far as I am able to ascertain, each patient is enjoying the best of health. At this writing I am treating ten cases of exophthalmic goitre, and their progress leads me to conclude that these will soon be added to the list of complete cures.

10. In addition to further devitalization of the patient, surgical

procedures unduly delay proper nonsurgical procedures, dangerously postponing the sought-for relief.

11. The scar and mutilation of the neck from thyroid surgery is embarrassing in after life.

Regarding the necessary duration of nonsurgical treatment, no definite rule can be formulated, as many factors must be taken into account. To quote from my article of the N. Y. Med. Jour., April 28, 1917: "Patients in whom treatment is begun quite early are cured in from six to eighteen months. Those whose treatment has been belated and have received no treatment for from one to three years are more difficult to manage, but these cases are far from incurable. With patient supervision of physician and strict obedience to orders by patient and caretakers, these late cases are as a rule entirely cured within from eighteen to twenty-four months. As implied, therefore, an early diagnosis is essential to early relief and cure. The case of six to nine months' duration is practically certain of complete cure within from six months to a year, one of a year's or eighteen months' duration requires more time for recovery. To generalize, it may be said that treatment to a successful outcome must continue about the same length of time as the previous duration of the disease. Many exceptions, however, occur, and occasionally after a year's treatment of a case of exophthalmic goitre it is necessary to continue a close observation of the patient for another six to twelve months before his or her discharge."

Lack of space precludes a detailed description of the nonsurgical treatment of Graves disease in this paper.

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## ABNORMAL UTERINE HEMORRHAGE IN YOUNG WOMEN

By ARCHIBALD McDONALD  
Gynecologist, Duluth Free Dispensary  
Duluth, Minn.

The significance of deviations from the normal menstrual habit in women under middle age frequently presents serious problems to the internist to whom these cases most often come. Seventy per cent. of women follow the usual twenty-eight day type with about four days of flowing, the remainder varying between twenty and

thirty days. At the extremes of ovarian activity; puberty and the menopause, there is sometimes irregularity in the rhythm, duration, and intensity of the flow. The individual menstrual habit is normally well established during the first year or so after puberty and is followed quite accurately throughout to the menopause. Menstrual blood normally remains uncoagulated: due to the alkaline cervical mucous secretion,<sup>(1)</sup> to the lack of fibrin ferment and fibrinogen<sup>(2)</sup>, to the presence of antithrombin in the endometrium,<sup>(3)</sup> or other substances which delay coagulation of plasma, demonstrated in the endometrium, ovary, and corpus luteum during menstruation.<sup>(4)</sup> The expulsion of clots is indication of excessive flow or retention of blood and represents pathological menstruation. Physicians have come to realize and patients must be instructed that: 1. Each woman sets her own standard or rhythm of menstruation in regard to: interval, duration, and in considerable degree to the character and type of pain, and intensity of flow. 2. Deviation from the accustomed type is pathological and must be explained.

These cases as presented to the physician fall into one of two rather distinct groups: 1. Those representing sudden deviation from a previously regular menstrual habit, possibly associated with amenorrhea, and suggesting pathology of early pregnancy. 2. Conditions of longer standing; Menorrhagia (profuse and prolonged menstrual bleeding) or Metrorrhagia (intermenstrual bleeding), either of which may be important: (a) as a symptom of serious lesion, new growth, or (b) as a cause of constitutional changes, anemia. Significant evidence to be noted includes: a period of amenorrhea five to eight weeks followed by atypical menstruation, sudden profuse flow with clots, irregular bleeding varying from scanty bloody discharge to profuse hemorrhage, prolonged flow, unusual pain especially unilateral, or the same conditions with no amenorrhea. Such deviation when sufficiently pronounced to bring the patient to her physician is evidence of general or local pathological change and must be explained.

Constitutional causes must be considered in some cases, but can usually be excluded by the history or examination. Change of climate from a warm to cool locality may be associated with amenorrhea followed by atypical flowing, most often, however, in women who have previously been irregular. Nervous influence, apprehen-

sion or fear of pregnancy often causes brief amenorrhea and subsequently a profuse atypical flow. Prolonged nervous strain may be associated either with amenorrhea, or in other cases, menorrhagia. With hysteria there is sometimes profuse bleeding with no apparent cause.<sup>(6)</sup> Pulmonary tuberculosis in young individuals is not infrequently associated with menorrhagia for a few periods to be followed by scanty flow and later amenorrhea.<sup>(6)</sup>

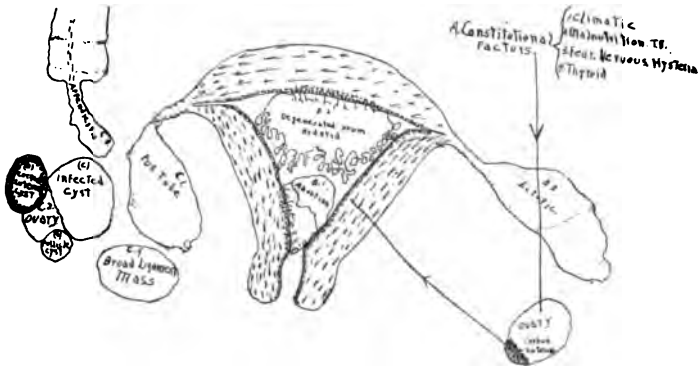


FIG. 1

Showing causes of sudden, atypical, bleeding.  
A. Constitutional. B. Pathology of early pregnancy.  
C. Pelvic Lesions. Possibly associated with pregnancy.

Pathological activity of certain remote glands of internal secretion has a more or less direct influence on menstruation, but the resulting deviations are usually of longer duration. Thyroid deficiency is associated with menorrhagia, which is improved by administration of gland extract.<sup>(7) (8)</sup> Menorrhagia is also observed in some forms of toxic, exophthalmic goiter. Conversely, I have notes of a case of menorrhagia, pelvic pain, and irritation, with nervous symptoms suggesting Grave's Disease of long standing, in which removal of a calcified ovarian tumor and sub-total hysterectomy relieved all symptoms. A few cases of adrenal tumors or hypertrophy result in menorrhagia due either to internal secretion or pelvic hyperemia. Pituitary lesions are most often associated with sexual dystrophy and amenorrhea.

Sudden abnormal bleeding in young women following an amenorrhea, however brief, is due to disturbance of early pregnancy till proven to the contrary. The most serious possibility is ectopic ges-

tation with impending rupture, and this must be eliminated by examination under anesthesia if necessary, or by exploratory laparotomy if there still be reasonable doubt. This diagnosis is made on a complex of: amenorrhea, pelvic pain, bleeding, and a tender mass lateral to the uterus, made out on bimanual examination. The picture may be more or less closely simulated by a number of other conditions. Those associated with pregnancy include: impending abortion, especially when accompanied by adnexal lesions; cysts, hydrosalpinx, or varicocele; abnormal implantation of the placenta, degeneration of the ovum, hydatidiform mole, or chorio-epithelioma, and all cause bleeding and pain.

In the absence of pregnancy there are several lesions which give a clinical picture easily confused with ectopic gestation and characterized most prominently by: atypical bleeding, unilateral pelvic pain and tenderness. 1. Mild exacerbation of chronic tubal infection or prolapse and torsion of a hydrosalpinx cause pelvic pain and atypical bleeding but no amenorrhea. 2. Ovarian Cysts. (a) Corpus Luteum cysts. <sup>(9)</sup> <sup>(10)</sup> Abnormal development or cyst formation in the corpus luteum causes delay in the menstrual period, enlargement of the uterus, and an atypical flow. The picture closely resembles ectopic. (b) Certain retention cysts due to atypical development of the graafian follicle, especially when these contain lutein cells and secretion, may also give a confusing symptom-complex. (c) Recent inflammation of small ovarian cysts with adhesions to pelvic structures and pelvic hyperemia may cause menorrhagia or, in a few cases, metrorrhagia, with lateral tenderness. These occur often with no history of recent pelvic disturbance, and may be secondary to remote septic conditions, tonsillitis. <sup>(11)</sup> <sup>(12)</sup> 3. Appendiceal inflammation usually of the subacute type with adhesions to pelvic structures may cause atypical uterine bleeding without amenorrhea. In such doubtful cases laparotomy is fully justified. 4. Small masses in the broad ligament; fibroids, cysts, or varicocele, rarely cause confusion except in the presence of impending abortion, when the mass made out on bimanual examination, together with enlargement of the uterus, pain and atypical bleeding may be mistaken for an ectopic.

Longstanding abnormal bleeding is of concern as a symptom of serious lesion, or as a cause of severe anemia and malnutrition. Metrorrhagia is always of pathological significance, whether con-

sisting simply of irregular bloody discharge or copious intermittent bleeding, especially when occurring at or near the menopause. The causes include: 1. Abnormal uterine contents, retained secundines of a remote pregnancy which is often unrecognized as such, or development of chorio-epithelioma, which is characterized by bleeding in 66% of cases.<sup>(13)</sup> 2. Lesions associated with congestion or



FIG. 11

Gross lesions causing Metrorrhagia by mechanical relations.

*History* not significant except sterility; 2-3.

*Recent history*, suggestion remote pregnancy, incomplete abortion, discharge.

*Pain*, not typical. *Bleeding*; intermittent, menorrhagia, sudden profuse or scanty bloody discharge. *General examination*, not characteristic, except secondary anemia, and malnutrition. *Local*, always indicated with metrorrhagia, must exclude cancer any age over 25. Enlarged uterus, friable cervix, speculum.

*Curetment* only to confirm diagnosis, no therapeutic value.

ulceration of endometrium: submucous fibroids, polyps, or cancer. Socalled "endometritis" is now excluded as a cause of uterine bleeding and almost as a pathological entity by the observations of Hitschmann and Adler, Novak, and others. Microscopic pictures described as forms of endometritis, are interpreted as stages of the menstrual cycle, dependent upon the periodic development of a graafian follicle and corpus luteum. Certain types of abnormal bleeding may be due to irregular development of a follicle but rarely extend over several periods. Examination together with visual inspection of the cervix gives the diagnosis only in case of marked enlargement of the uterus, cervical polyp, or cancer, and is by no means final. Curetage, so often advised for these cases, should be done for diagnosis only, together with microscopic exam-

ination of curettings. It will be of therapeutic value only in case of retained secundines, or in some instances where a polyp can be removed at the same sitting. It was found of value in less than 10 of over 600 cases.<sup>(14)</sup> The repeated curetments to which these patients are often subjected for persistent bleeding of unknown origin are unjustifiable. I have notes of one patient, which is by no means rare in the experience of others, for whom at least four curetments had been done but with no diagnosis. The bleeding recurred promptly each time and finally a sub-total hysterectomy was done, when examination showed a small polyp with cancer in the fundus. Examination of curettings must be done by an expert since the microscopic picture of different stages of the menstrual cycle may be confusing, and a thorough curetage may miss a small area of cancer in the fundus. In certain cases where the diagnosis is still in doubt, exploratory hysterotomy as described by Russell,<sup>(15)</sup> and Deaver,<sup>(16)</sup> is justified.

The importance of copious menstrual bleeding is measured not by the amount of blood lost, but by the effects on the patient, i. e., when it causes progressive anemia and malnutrition which is not overcome before the next period. Results may be extreme and overshadow the menorrhagia, the existence of which is brought out only by a careful history. In many instances periodicity is lost and flowing recurs every two or three weeks but with no intervening flow. In protracted cases the resulting anemia causes blood changes which in themselves predispose to menorrhagia. A safe working diagnosis is the first essential. The type of bleeding, history, and local examination, is usually sufficient to exclude gross lesions of the uterus. Large ovarian tumors which are easily made out on examination are not often associated with bleeding, but small fibrous ovaries with no palpable tumor may cause or at least be associated with marked metrorrhagia.<sup>(17)</sup>

The terms "Essential Hemorrhage" or "Myopathica Hemorrhagica" apply only to cases where gross lesions of the uterus or adnexa have been excluded and the cause of the bleeding is referred to microscopic changes in the endometrium or myometrium, or to pathology of the internal secretions.

In order to discuss pathological menstrual bleeding it is necessary to accept a working hypothesis of the phenomena associated with and

governing the normal process. This is all dependent upon the secretions or hormones from the ovary, particularly those connected with the maturation of the graafian follicle and development of the corpus luteum, since it occurs only during the period while these are active, i. e., between puberty and the menopause. The process ceases after removal of the ovaries or destruction of the follicles by Roentgen ray, but persists after all nerve supply has been severed, or there

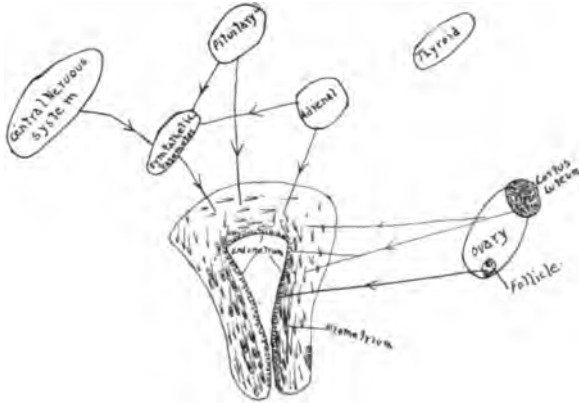


FIG. III

Showing possible factors in "essential hemorrhage."

remains only a small portion of one ovary and that, possibly transplanted. Therefore the stimulus is chemical and is independent of nerve supply. The periodicity is difficult to explain, not being influenced by removal of one half or two thirds of the total ovarian tissue, and therefore a corresponding proportion of the graafian follicles. We assume that when the ripening of one follicle has reached a certain stage, development of others is inhibited till the cycle is complete. Since there is an infinite number of ova, the periodic process is repeated as long as there is ovarian tissue so situated that ova can mature and reach the surface. The relationship between the cycle in the follicle and that in the uterus has been worked out in many particulars.<sup>(18) (19) (20)</sup> Changes in the uterus consist of hypertrophy of the endometrium and underlying stroma, increased vascular supply with dilated vessels, all in preparation for fertilization and implantation of the recently matured ovum. When impregnation does not take place, the congestion is relieved by men-



strual bleeding which consists of diapedesis of blood from engorged vessels of the endometrium.<sup>(21)</sup>

The exact relationship of the corpus luteum to menstrual bleeding is difficult to determine. It develops in the ruptured follicle from cells of the granulosa and has a characteristic microscopic structure. Chemical studies of extracts<sup>(22)</sup> indicate special function, and clinical observation<sup>(23)</sup> demonstrates an internal secretion of great importance. It is vital in the conservation of early pregnancy demonstrated in animals,<sup>(24)</sup> and in the human serves to maintain stability of the implanted ovum, either by increasing uterine hypertrophy and hyperemia with changes in the endometrium, or by inhibiting uterine contraction and bleeding. The corpus luteum of pregnancy differs in development, duration, and structure, from that of the normal menstrual cycle. Cysts or degeneration of the corpus luteum are associated with abnormal pregnancy or atypical bleeding. We assume that in case of pregnancy, hypertrophy of the corpus luteum follows and in turn through the influence of its secretion, changes in the uterus and inhibition of other follicles. In the absence of pregnancy there is atrophy of the corpus luteum, uterine bleeding, completion of the menstrual cycle, and development of another ovum follows in regular order. The question arises, does menstruation occur as the result of hormones from the corpus luteum directly, or rather does it follow atrophy of this structure in the absence of pregnancy? Clinically corpus luteum extract seems to reinforce deficient ovarian tissue but is not sufficient to initiate menstruation after the natural or induced menopause.

The methods of normal control or limitation of menstrual bleeding presents several possible explanations. Cessation of hemorrhage represents the final stage in the cycle of development of the graafian follicle with the decline of certain internal secretions which caused the premenstrual uterine changes; hypertrophy and hyperemia of the endometrium, presence of anti-thrombin, etc. Ovarian secretions are active in maintaining general tone of myometrium, but otherwise no active agent limiting uterine bleeding has been demonstrated from the ovary. Normal contraction of uterine muscle controls puerperal hemorrhage by constricting vessels of the myometrium. On the same basis it is fair to assume that similar activity of uterine muscle, associated with the decline of the pre-

menstrual stimuli, is sufficient to control menstrual bleeding. Normal physiological activity of the myometrium is to a considerable degree under control of the secretions of special glands; adrenal and pituitary, which are carried in the circulation and have been shown experimentally to stimulate uterine contractions.<sup>(25)</sup> It is easy to understand that normal functional activity of the uterus may be impaired by a number of conditions with no gross lesions of that organ. Such loss of physiological tone may be due to: 1. Subinvolution especially when resulting from rapidly repeated pregnancies. 2. Chronic passive congestion with malposition. 3. Inflammatory changes with infiltration of the myometrium. 4. Premature or excessive senile fibrous degeneration and arteriosclerosis, especially when associated with increased ovarian stimulus. A review of recent literature includes many excellent articles, each of which attempts to charge excessive bleeding to some particular factor. One is forced to the conclusion, however, that no particular one can be accepted to the exclusion of others. As a working hypothesis we can assume that there is a lack of balance between the ovarian hormones which initiate the premenstrual changes on one hand, and the functional activity of the myometrium and controlling factors on the other. But always with the realization that intensive examination or exploratory laparotomy will demonstrate an anatomical cause in some cases.

Therapeutic measures include: 1. Gland extracts and general treatment. 2. Roentgen Ray and Radium. 3. Exploratory laparotomy and hysterectomy.

Specific therapy is said to give striking results in some cases of thyroid deficiency, but the use of other gland extracts is less definite. The action of corpus luteum and ovarian preparations has not been explained, and has been recommended for menorrhagia, but most often for scanty menstruation or amenorrhea. Constitutional measures: rest and tonics, are too often of only temporary value, recurrence being common. Unrecognized malignant growths may progress during such treatment.

2. The Roentgen ray and more recently radium, is claimed to effectually control bleeding due to fibroids or "Essential Hemorrhage" though the exact mode of action is in dispute. One group of workers<sup>(25)</sup> claim that the action is due primarily to destruction of the

follicle apparatus of the ovary and an induced menopause. Others<sup>(24)</sup> describe the chief action on the uterus or tumor through sclerosis of vessels and consequent limitation of bleeding, since this may result without causing the menopause and that post-climacteric bleeding from fibroids is equally well controlled. Successful treatment in young women, however, usually results in an objectionable premature menopause and causes undesirable effects in other cases, while the necessary time and expense make the method prohibitive in many instances. The possibility of overlooking malignant growths and continuing treatment only long enough to control hemorrhage is a real danger. Contra-indications include: inflammation of adnexa, rapidly growing fibroids, or operable malignant disease.

3. Exploratory laparotomy with hysterectomy as indicated presents many advantages. It permits an accurate diagnosis and appropriate treatment of pathology without delay or uncertainty. In the absence of gross lesions, subtotal hysterectomy effectually controls bleeding without inducing the menopause. In reasonably selected cases the operative risk is slight, the invalidism is short and results are permanent. Rapidly repeated pregnancies or abortions are important etiological factors in many cases and the resulting sterility is an added protection. In many cases surgical correction of malposition, and tubal sterilization when indicated, will be sufficient, but recurrence may demand hysterectomy later. Finally patient and physician can be assured that there is no danger from overlooked malignant disease.

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## THE DIAGNOSIS OF TRAUMATIC NEUROSIS IN RELATION TO LITIGATION AND COMPENSATION.

BY TOM A WILLIAMS

Neurologist to Freedmen's Hospital, Howard University; For. Corresp.  
M. Neurol. Soc. of Paris, etc.

Washington, D. C.

The true nature of the reaction of persons complaining of neuroticism after an accident is psychological. In this question the important problem is to differentiate between the person who is deliberately trying to deceive some one for material gain, and the patient not trying to deceive, but who wishes to be sick because of the belief that he ought to be.

Now, these two situations, although morally different, are not distinguishable by neurological methods. The reactions are similar; the motives differ, only by contingent evidence is detection possible. I cannot here enter at length into the considerations to be borne in mind in such cases. Those interested must consult my article on Traumatic Neurosis, Am. Jour. Med. Soc., 1914, or on Occupational

Neurosis, before International Congress of Medicine, London, 1913, Trans. Neurological Section. All one can do is to find out whether or not we are dealing with some physical condition or with some psychical situation which exists in the patient's mind as to what he considers a grievance. If he can be shown that he has no grievance because he is sick only because of his point of view, he can be cured, and of course will then forego the litigation for damages. That can be done, as I can personally vouch by examples.

But we must not forget that even a person who has some physical damage can be made not to feel pain if he is distracted; and I have seen gross mistakes made in saying that patients were neurotics because they ceased to feel pain when they were distracted. If the distraction is serious enough, the pain of a very severe blow will not be felt, as any one knows who has played football, when a severe blow on the shin is not felt at all, because of the distraction by the excitement of the game. So we must not draw too hurried a conclusion about a patient in whom our examination creates a modification of the sensibility.

My experience as a neurologist is not the same as that of those who say that most functional paralyses are spastic. Along with Babinski and other authorities, I find the majority of psychogenetic paralyses to be flaccid. But diagnostically it does not matter whether a paralysis is flaccid or spastic in order to determine whether it arises or not from some impairment of the neuronal arc. The reflexes are an infallible guide if observed by one who knows, and when correctly interpreted. Any neurologist should make a diagnosis infallibly, and every surgeon might learn a few of the simple signs. For instance, a simple sign for organic paralysis of the arm is to put the forearm up erect on a table. If the neurones are intact, it stays that way; but if there is spastic or flaccid paralysis it will fall, because of failure of innervation through the pyramidal tract. In a psychogenetic paralysis the wrist does not fall, because the patient has no inkling that it should fall.

The best way to handle cases of neurotic accident, however, is through a medical referee. For the patient is rarely going to think the railroad surgeon is not prejudiced in favor of the company. His own doctor is often in sympathy with the patient, and is apt to reinforce his grievance. I have had a number of cases referred to

me in this way, and the patient can generally be satisfied by my interpretation. If the corporation can be convinced, too, the case does not go to trial. Sometimes the patient cannot be satisfied. In the Hill case I could not beforehand convince the patient; but his idea that he had spinal disease was disproved at the trial by his walking and doing exactly opposite to what he should have done, which the jury was shown by testimony in court. See Report in New Orleans Med. Jour., St. Paul Med. Jour., 1915; also in Jour. Criminal Law, Jan., 1917.

However, if there is introduced other testimony, however authoritative, of some one not versed in neurology, the case is apt to be lost, because confusion will be raised in the minds of the jury. I have been in two such cases—one in Washington, one in Richmond—where a witness unversed in modern neurology was introduced so that the cases were lost on account of the use of meaningless technical terms which only confused the jury.

Even where the cause is purely psychological and a rational, simple explanation has sufficed to cure the patient, the corporation should compensate for loss of time already endured. Even though the recompense is small, the matter is thus usually adjusted satisfactorily. There is pending a case in Washington which the lawyers on both sides have asked me to settle.\* Railroad companies have told me they would rather I was not connected with them, because I could be of more use to justice as an independent neurologist.

I believe that every neurologist can assist both the patient and the railroad surgeon by impressing upon the former, when there is no physical cause, how he can get away from feeling as he does. But that can only be done by a person thoroughly conversant with the diagnostic technic of neurology and with modern psychopathology and with sound psychotherapeutic procedure.

\*This case has since been settled on the basis of my report, but unfortunately from the point of view of the corporation, the patient proved to be more sick than her doctor had supposed, and in consequence the indemnity was increased, because of my report, on demand of the plaintiff's attorneys.

✓ METHODS OF CHARTING PHYSICAL FINDINGS IN  
LUNG DISEASES

By ERNEST ZUEBLIN

Medical Director, Cincinnati Tuberculosis Sanitarium; Associate Professor  
of Medicine, University of Cincinnati  
Cincinnati, Ohio

Among the clinical records the proper method of charting our physical findings in a concise and comprehensible way has still to be found. Many attempts have been made to solve this problem and still, in reading the physical signs such as used in the clinical description of cases, we get easily lost among the various expressions used to describe and convey a clear understanding of a physical status. Should it not be possible to find a proper way of expression, which by its flexibility and adaptability gives a quick understanding of the principal physical signs? Just as shorthand writing has gained popularity among all the professions, the aim is worth while to work for a common understanding among medical men for a code of signs, easily reproduced in printing!

Although I do not claim that the method described below is the only and ideal one, I do not hesitate to bring it to the knowledge of the profession, since in my experience in practice and medical teaching it has proven of great value to me to grasp at a glance the chief chest findings, to state at a glance whether the patient's condition has changed for the better or for the worse.

In our aim to determine the extent of a lesion, we are no more satisfied with the simple statement that such and such a physical sign is present, but as in analytical geometry we try to project the position of a point with regard to its ordinate and abscisse on one plane, so in physical diagnosis we project our signs arising from the depth of the thoracic cavity on the paper, indicating exactly the spot where it has been found and where it can be controlled by the successive examination. The landmarks used are, of course, the different regions of the chest, the dividing horizontal and vertical lines, the intercostal spaces anteriorly, the interspinous notches posteriorly. The adoption of the code, of course, requires some exercise, some study, until we have become thoroughly familiar with that code of abbreviations, but once acquired it will save a lot of time in avoiding confusion and erroneous interpretation.

Before giving a practical example, I intend to give a short list of the main physical signs with their abbreviations as follows: R.—right, l.—left, L.—Lung, A.—apex, B.—base, up.—upper, md—middle, lw—lower, ant.—anterior, post—posterior, ax.—axillary, mam—mamillary, scap—scapular, cl—clavicular, Isp—Interspace 1, 2, d. first, second, etc.; Rb. I.—first rib, etc.; Spcp.—spinal cervical process, Sppd.—spinal dorsal process, Ispnd—interspinous dorsal notch, F—fossa, IclF.—infraclavicular fossa, ScfF.—supraclavicular fossa, SscapF.—suprascapular fossa, IscapF.—infrascapular fossa, ItscapR.—Interscapular region, Agscap.—angle of scapula, MSL—midsternal line, PstL.—parasternal line, MCIL.—midclavicular line, MML.—Mammary line, AxL.—axillary line, aAxL.—anterior axillary line, mdAxL.—middle axillary line, postAxL.—posterior axillary line, ScapL.—scapular line, mdScapL.—mid scapular line, retr.—retracted, protr.—protruding, Ex.—expansion, — diminished, + increased, Rex.—respiratory expansion, TF.—Tactile fremitus, VF.—vocal fremitus, Ppt.—Palpation, P.—percussion, —1 slightly diminished, —2 dull, —3 flat, +1 slightly increased, +2 marked increase, +3 hyperresonant, Pt.—tympanitic percussion, Pcp.—cracked pot percussion, Pp.—palpatory percussion, —1 slightly diminished, —2 markedly diminished, Pn.—normal percussion, Resp.—respiration, + or — increased or decreased, Respp.—respiration puerile, Respv.—vesicular respiration, Respb.—bronchial respiration, Resp. ind.—indefinite respiration, Respabs.—absent respiration, Insp.—inspiration, Insp. gr.—inspiration granular, Inspw.—inspiration wavy, Inspcw.—inspiration cogwheel, Inspirr.—inspiration interrupted, Exp.—expiration, h.—harsh, Exp.  $\searrow$ —low pitched, Exp.  $\nearrow$ —high pitched, Exp.  $\circ$ —expiration prolonged, Resp.  $1/2$ —inspiration 1, expiration 2, or expiration double length from inspiration;  $3/1$ —inspiration 3, expiration 1, or one-third of inspiration; Expb.—expiration bronchial, Expt.—expiration tubular, Expta.—expiration tubular amphoric, Expmus.—expiration musical, Rl.—rales, Rlcr.—crepitant rales, Rlmc.—mucous rales, Rlfmc.—fine mucous rales, Rlcmc.—coarse mucous rales, Rlss.—sibilant rales, Rlmdmc.—medium-sized mucous rales, Rlmet.—metallic rales, diff.—diffuse, sct.—scant,  $\rightarrow$ —as far as, Rlmus.—musical rales, Rlcs.—consonant rales, Rlgg.—gurgling rales, Rlac.—rales after cough, Fr.—friction rub, Bph.—bronchophony, with intensity 1, 2, 3, i.e.,



slight, medium intensity, marked; Pec. 1, 2, 3, slight, marked, intense pectoriloquy, Rlq.—raloquy an increase in intensity of rales whilst patient is whispering, Egph.—egophony, Hipp. S.—Hippocrat succussion, CS.—coin sound, PtS.—post tussis suction.

In an inquiry among practising physicians as to the ways adopted of marking physical signs, it struck me that almost every one of them had adopted a system of his own, which markings, notwithstanding the brevity and clearness, required a translation into the more classic terms adopted for physical diagnosis. In the various text-books on physical diagnosis (Sahli, Klebs, Hutchison and Rainey, Pottengers, and others) a graphic sign method has been used, which signs in printing require special types and, according to my personal experience, publishers are not very much in favor of carrying such types in stock, just on account of the lack of unity among the medical profession.

The practical application of the shorthand system may be illustrated by the following example:

r. L. ant. A. retr. 2 → 2 Isp. Rex. — 2.	Right lung over apex considerable retraction, extending as far as second interspace, with marked lack in respiratory expansion.
VF. + 2 → 2 Isp.	Marked increase of vocal fremitus extending to second intercostal space. Diminished percussion sound from apex to second interspace.
Pp. — 2 → 3 Isp. P. — 2 → 2 Isp.	Marked diminution of sound on palpatory percussion extending from apex to third intercostal space.
Insp. gr. → 2 Isp. Exp. ∘ h. → 2 Isp.	Granular inspiration and prolonged harsh expiration as far as second intercostal space.

- Rl. cr. dif. 3 i, Rl. mdmc. 3 ex. → 3 Isp. Diffuse crepitant rales during inspiration and diffuse medium-sized mucous rales during expiration extending from right apex toward the third interspace.
- Bph. 1 → 3 Isp. Pec. 3 → 2 Isp. Slight bronchophony in third interspace, marked pectoriloquy over right apex extending to first interspace.

FOR THE RIGHT APEX POSTERIORLY

- R. L. post. A. retr. 2 → SscapF. Rex. — 2. Marked retraction and marked lack of respiratory expansion over right apex posteriorly and suprascapular fossa.
- VF. + 2 → Sppd. 3. Marked increase of vocal fremitus over right apex posteriorly as far as third dorsal interspinous process.
- P. — 2 → 3 Sppd. Diminished percussion sound over right apex extending to third dorsal interspinous process.
- Pp. — 2 → 4 Ispdn. Marked impaired percussion sound with palpatory percussion extending from apex to fourth interspinous dorsal notch.
- Insp. gr. Rlcr. 3 i → A. → Spp. 3 Granular inspiration, numerous crepitant rales during inspiration extending from apex to the third dorsal process.
- Exp. ∞ h ↗ Rl. mc. 3 ex A. → 3 Sppd. Prolonged harsh expiration, high pitched, with many mucous rales during expiration extending from apex to third dorsal process.
- Pec. 1 — 2 → 3 Sppd. Marked increased pectoriloquy over right apex posteriorly extending to the third spinous dorsal process.

Bph. 1 → 4 Ispdn.	Slight bronchophony over right apex extending as far as fourth interspinous dorsal notch.
Rexp. $\frac{1}{8}$ " ← $\frac{5}{8}$ " → $\frac{3}{8}$ ".	Respiratory expansion of apex on ordinary respiration and during deep inspiration toward median line amounts to $\frac{1}{8}$ of an inch, whilst the expansion toward the acromion is $\frac{3}{8}$ of an inch. The expansion under normal breathing extends over an area of 1 inch and $\frac{5}{8}$

As for the physical findings of the basis of the right lung, those of the left lung, apices and bases included, they are recorded in a similar way. It will hardly be necessary to carry the example further. It may suffice to call the attention to the abbreviations which express in a few lines all the details desired for comparison and diagnosis. Even in the attempt of describing as briefly as possible the physical findings in the usual terminology, many lines are required for the description and still an attentive reader may become lost among the many lengthy details.

For the lung specialist, where every intercostal space, in front, in the back, in the axillary regions, have to be searched for the presence or absence of physical data, we have found the following scheme (Table 1) very useful, based upon the direction of the vertical dividing lines, such as parasternal, midclavicular, anterior, middle, posterior axillary, the midscapular and scapular lines. This is done not only for the purpose of obtaining a clear idea of the extent of pulmonary involvement for diagnosis, prognosis and the changes secured by medical and physical treatment, but also in view of a comparison of the physical data with the readings of stereoscopic X-ray plates.

At a later opportunity I will have the pleasure to show in which way physical findings and X-ray plates furnish us a proper way in obtaining a more accurate understanding of normal and pathological chest conditions, which better stand the criticism and revelations of the pathologist.

[illegible]

## TABLE 1

Pp.—Palpatory percussion; Insp.—Inspiration; Rl.—râles; cr.—crepitant; i.—inspiratory; e.—expiratory; h.—harsh; Exp.—Expiration;  $\frac{1}{2}$ —length of expiration one half of inspiration; tb.—tubular; — prolonged; W.V.—whispered voice; Bph.—Bronchophony; Pect.—Pectoriloquy; Pect. 1—slight; 2—marked; — minus; + plus; increased; M increased pitch.

## TOPOGRAPHIC PERCUSSION OF THE CHEST COMPARED WITH POST-MORTEM FINDINGS

By ERNEST ZUEBLIN

Associate Professor of Medicine, University of Cincinnati; Resident Medical  
Director of the Municipal Tuberculosis Sanitarium of Cincinnati  
Cincinnati, Ohio

Physical diagnosis in chest diseases is given a leading rôle in the curriculum of the medical student. In approaching this subject, the beginner is reminded of the classic saying: "Ars longa, vita brevis," art is long lasting, whilst life is short. The medical student may take a long while before acquiring the proper translation of physical signs into the pathological changes, which constitute the cause of the morbid manifestations. For some of the medical men this primary stage of hesitation is never overcome, physical diagnosis remaining for them "a noli me tangere." Intent study and much practical experience are the means by which these initial difficulties are conquered leading up to perfection in the application of the methods of physical diagnosis. Once we have graduated, the standpoint of our former teachers is still reflected in our work, who may remain fascinated by exclusive and further reaching advantages attributed to percussion or auscultation alone, whilst favoring the one method the other may be neglected. It will happen that our own technic will follow its own course, but we will gladly adopt all the different steps which will secure the desired information about the patient's condition. Just as in daily life we enjoy the sunlight as such, without a thought of using alone the heat, the light and ultraviolet energy exclusively, in physical diagnosis we resort to all the means known to acquire the proper information.

In chest diseases we are no more satisfied to ascertain the nature of the disease threatening human health, our scientific interest leads us further to ascertain the *extent of pathological lesions*, with the aim to gain besides the diagnosis the proper information for the prognosis and treatment of the case. Refinement of our technic, additional means of diagnosis, X-ray examinations and, last but not least, post-mortem examinations must furnish us the proper understanding of the pathological changes.

In former editions of books on physical diagnosis we find the routine methods well explained and taught, but the more delicate procedures of palpatory, topographic, plessimeter and direct percussion are better appreciated in recent text-books on the subject. In addition to the pitch, quality of the percussion note, we learn to utilize the variations of tissue resistance encountered over normal and diseased areas. The palpatory and tactile impression recorded by the finger applied over the diseased area means a further point of consideration in the final summing up of our findings. The immediate comparison of percussion findings with the post-mortem changes—so far to my knowledge—has not yet been practiced in many clinical institutions. Scepticism of the reliability of our methods may be one of the reasons why we slowly adopt new methods of investigation besides those taught in our schools. Personal curiosity into the problem was one of the reasons of presenting the present study, dealing with our tubercular patients. In exposing the facts obtained by physical examination—chiefly by palpatory percussion—and comparing them with the report of the pathologist, it is not my purpose to give preference to one single method before all others. I am fully convinced that a physical diagnosis will only be satisfactory if in our technic we resort to all the means, which step by step must secure us the desirable information. In doing so from our personal experience we cannot eliminate instances, where all our methods failed to give us a clear understanding of the factors at cause of the morbid symptoms and in which instances the post-mortem examination furnished us the only explanation of symptoms unrevealable by other means of diagnosis.

The following cases, among many others at hand, will demonstrate the pathological anatomical explanation to the abnormal palpatory and percutory findings:

Case I. B. D., colored, age 34, admitted on 2-24th-17, died 2-27th-17, in very weak condition when admitted, semidelirious, impossible to obtain any etiological data, high temperature, 104°, pulse 140, respiration varying from 28 to 40 pro minute. Physical diagnosis unsatisfactory. *Right lung anteriorly* presented impaired percussion sound from apex and inclusive fourth intercostal space, amphoric breathing in right supraclavicular fossa, prolonged in-

spiration, fine subcrepitant rales in region between first and third intercostal space, with suggestion of cavity in 2d and 3d interspace, outside of midclavicular line. In 5th interspace prolonged inspiration and fine rales. Right lung *posteriorly* with ordinary percussion dulness as far as level of fourth interspinous notch. Impaired percussion sound throughout right base. Auscultatory changes, bronchial inspiration, fine crepitant rales from apex downward to level of fourth interspinous notch, prolonged inspiration downward all over right basis.

The percutory findings over the *left lung anteriorly*—downward from apex and including third interspace—impaired percussion sound, tympany over left basis anteriorly. On auscultation bronchial breathing from apex to 2d interspace, prolonged inspiration in 3d and 4th interspace, absent breathing over remaining left basis. *Posteriorly* the dulness extended from the left apex to the level of the 5th interspinous notch, and below this area impaired percussion sound existed all over the left basis. On auscultation prolonged expiration and fine subcrepitant rales from apex to level of 5th interspinous notch, below this level all over the left basis breath sounds were completely absent. The patient's sputum, mucopurulent in character, was simply loaded with beaded tubercle bacilli.

Immediately after permission of a post-mortem examination was obtained, post-mortem rigidity being noticeable, the body was examined with ordinary and *slight threshold* percussion, in order to control the auditive and tactile impression by the visual and palpatory changes, once the thoracic cavity being opened. Below I shall give only briefly the results and then give the protocol of our pathologist. Over the *right apex anteriorly* with palpatory percussion the sound is markedly *impaired*, and extends to the lower border of the *second rib*, the lower half of the 2d interspace is resonant and resilient as far as the 3d interspace, below this level impaired resonance and resistance are mapped out, involving the right basis anteriorly. *Posteriorly* impaired percussion sound, marked resistance is felt over the *right apex*, namely the *right suprascapular fossa*, and in the *interscapular space*, as low as the level of the 4th interspinous notch, corresponding to the upper third of the median border of the right scapula. Below this level a

*small* triangular space of normal resonance is mapped out, the hypotenuse of said triangle runs parallel to the 6th rib. Right below we find again a *triangular area* of marked *dulness*, extending from the 4th to the 6th *interspinous notch*, the basis extending to the lower third of the median scapular border. The next zone immediately below reveals a quadrangular area of hyperresonance, extending from the 6th to the 8th interspinous notch, its lateral border being formed by a convex line, slightly outside of the midscapular line, anteriorly of which a zone of dulness is encountered which extends toward the right axilla, merging into a dulness marked anteriorly and extending into the zone of abnormal dulness marked in the 4th and 5th interspace, anteriorly. Posteriorly, from the 8th interspinous notch, involving the remaining right basis, marked dulness is noticed. On the *left side anteriorly* the following changes are recorded: Over the *left apex*, in mapping out Kroenig's areas of resonance, a very narrow strip is made out, which on indirect and strong percussion gives hyperresonance. Below the clavicle the percussion note is impaired as far as the *lower border* of the 2d rib. The second and third interspace anteriorly, outside of midclavicular line, give a *hyperresonant* percussion sound, low pitched in character. The *relative cardiac dulness* is encountered inside of the midclavicular line  $3\frac{1}{4}$ " from the midsternal line just below the border of the 5th rib, the right border of the relative cardiac dulness is about  $\frac{1}{4}$  of an inch inside the right sternal border. With strong indirect percussion the absolute cardiac dulness comprises a small area, beginning in the 4th interspace, right along the left sternal border and extending into the fifth interspace, it reaches not more than  $\frac{3}{4}$  of an inch outside of the left sternal border. Over the left *basis anteriorly* an area of dulness presents itself, the upper borders of which spreading in an ascending line, from the upper border of the 5th rib in the midclavicular line and following the direction of the upper border of the 4th rib into the left axilla. *Posteriorly*, starting with the left suprascapular fossa, which is equally depressed, a mixed sound of tympany and dulness is found. The latter sound quality is spreading to a horizontal line, comprising the *lower border* of the *scapular spine* and above the middle between the 2d and 3d interspinous notch. Right below this limit, in the *interscapular*



region, an area of resonance exists, adjoining a zone of *dulness* and *increased resistance* follows, which is triangular in shape, extends laterally from the *4th* and *5th interspinous notch*, the upper border of which is parallel to the direction of the 6th rib. The next and immediately adjoining area of hyperresonance comprising the lower third of the scapula and the infrascapular region as far as the 8th interspinous notch toward the midline, extending laterally to the posterior axillary line. Beyond this area the encountered dulness merges into that found arising from the Traubes space, mentioned anteriorly. The left basis of the lung is found dull on direct percussion and slight percussion, namely, below the 8th and 9th interspinous notch.

After these preliminary markings were mapped out, the thoracic cavity was opened in following the routine technic, without disturbing the genuine topography of the organs, simply removing the sternum the following changes were revealed:

No free fluid in the pleural cavities. The *left lung* presented dense adhesions at the *apex* of the upper lobe anteriorly, the lowest of which binding down the left upper lobe, following the lower border of the *second rib* past the left midclavicular line. The second and third interspace are found free from adhesions, but on section a fairly sized *cavity* corresponding to the lingual trunk and located in the *third interspace*, outside of the midclavicular line, intersected by the midclavicular line is found. Extending from the *5th interspace* toward the *axilla* there exist *dense adhesions*, which are located in the *fourth interspace* and extending toward the posterior aspect of the pleural cavity. *Anteriorly* the *left apex* is studded by numerous *coarse nodules* of tuberculosis, which extend as far as the *lower border* of the *second rib*. *Posteriorly* the apex of the left lung presents numerous *nodules* on the surface, extending to the fourth and fifth interspace or the *third interspinous notch* and on section numerous *cavities* are found, whilst the *basis* of the left upper lobe reveals a heavy *bronchopneumonia*. At the *apex* of the *left lower lobe*, corresponding to the *5th interspace*, or the *fourth interspinous notch*, heavy *infiltration*, with *cavitation* extending about one inch downward, are found. In the 7th, 8th interspace there are no adhesions, the pulmonary parenchyma presents a tuberculous bronchopneumonia. From the 9th interspace down

to the left basis *dense adhesions* are encountered, which findings correspond well with the dulness mentioned on percussion.

The RIGHT LUNG *anteriorly* presented atelectasis of the upper apex, dense adhesions along the lower border of the first rib, diffuse palpable nodules which extend as far as the *upper border* of the *third rib* and passing the right midclavicular line they extend toward the right axilla. The rest of the lower part of the upper right lobe, and also of the middle lobe, show *bronchopneumonia* with areas of pulmonary edema. From the right axilla spreading posteriorly toward the *posterior axillary* line numerous tubercular *nodules* are found. *Posteriorly* the *upper lobe* reveals tubercular infiltration, which process extends to the *fourth interspinous notch*, or the *5th interspace*. Occupying the *middle third* of the scapula, the apex of the lower lobe is infiltrated by large nodules, and anteriorly to them the enlarged bronchial glands are encountered. Below this area corresponding to the *middle third* of this lobe a few discrete *nodules* of tuberculosis are found scattered in *edematous pulmonary* parenchyma. The *lower third* of the right basis all the way downward from the 7th interspinous notch, or the 9th and 10th interspace, *dense adhesions* along the ribs are found. So far the lung findings on this case, and in regard to the percutory findings and the tactile impressions made, before the post-mortem was started, the physical findings tally well with the post-mortem control. Where we have cavities buried in connective tissue and in pleural adhesions, it is difficult to ascertain their presence by the palpatory percussion on the post-mortem table. If there are no pleural adhesions, then we may ascertain them by the quality of the percussion sound, as in this instance in the lingual trunk in the third interspace anteriorly. If we encounter adhesions and tubercular nodes, as in the left upper lobe anteriorly, it means a hard guess to determine the exact proportion of each one of the pathological processes. Although the examination of the back means a difficult task, on account of the bony structures—scapulæ, ribs and the heavy muscular layer—it is interesting to find the various zones of different percussion sound verified by the post-mortem. No more satisfied to ascertain that there is a tuberculous infection, our interest is directed toward the recognition of the extent of the pathological involvement. It seems possible by care-

ful examination to differentiate areas of nodular infiltration from those of a bronchopneumonic process. The impression is gained from a careful study of stereoscopic X-ray plates that, at a certain stage of the disease, the apices of the lower lobes may be involved before the entire destruction of the upper lobe has taken place and, clinically the prognosis of such cases, where the destruction has gained access to the lower lobes, the prognosis changes considerably with the onset of such an event. Heavy infiltration, or even tubercular nodes, will be suggested by the findings of palpatory percussion, particularly such a suspicion is strengthened when auscultatory signs of harsh breathing may be present with fine crepitant rales, but frequently with bronchophony or pectoriloquy are found in vivo. By close attention to the existence of the *interscapular triangle*, located at the level of the middle third of the scapula in hundreds of medium-advanced cases, we could ascertain its presence. In the above case, however, one reserve may be made, that in some cases such a triangle may be associated with enlarged hilus glands. Unless the glands would be chiefly situated in the posterior mediastinum, an intervening layer of normal pulmonary parenchyma would diminish the palpatory impression. The possibility of muscular contraction and spasm in vivo might interfere, but in cadavers where the post-mortem is carried out before any sign of rigor mortis has occurred the muscular contraction can be discarded as a disturbing factor.

Case II. C. H., age 47, white male, admitted on 3-14th, suffering from pulmonary tuberculosis for the past 4 months, complicated by tubercular enteritis for the past month, died on 3-21st, autopsied a few hours P.M. Immediately preceding the post-mortem the chest was examined by slight palpatory percussion, the following data were obtained: Impaired percussion sound over *right apex* extending to *second interspace* and past the midclavicular line involving the *3d intercostal space* of the axillary region. Only two-thirds of the third interspace present a normal percussion sound, the *fourth interspace* being substituted by an area of *dulness* which extends from the right sternal border to the *axilla*, merging into the percutory density which spreads from the second and third interspace into the axilla. Posteriorly the *entire supra-infra* and *interscapular region* to the right from the spinal column is dull as far as the *8th*

*rib* and the *angle* of the *right scapula*. The 8th and 9th interspace are found *resonant* and of normal resiliency. Beginning with the *upper border* of the *tenth rib* and extending to the *base* and into the *right axilla*, a zone of *impaired* percussion sound is made out. The percussion of the *left lung* anteriorly shows over the apex, the *inner half* of the *first interspace*, *dulness*. The *outer half* of the *first interspace* gives a mixture of *dulness* with *tympany*—pumpkin sound. Following the direction of the left midclavicular line, and extending over the *second*, the *third*, *fourth* and *fifth* interspace and involving the *entire axilla* toward the posterior axillary line, a *diffuse dulness* is encountered. Posteriorly the *left apex* presents a *dulness* as far as the *fourth interspace* in the *interscapular region*. The 5th interspace below is normal, but below the 5th interspace a *triangle* of *dulness* is made out, which extends from the median line laterally to the angle of the left scapula. Below this *triangle* a very *small area* of *hyperresonance* hardly wider than one inch is made out, which extends to the *left axilla*. Beginning with the *upper border* of the *9th rib*, close to the median line, intersecting the *8th interspace*, a zone of *dulness* seems to occupy the *entire remaining left basis*. The cardiac *dulness* extends from the midsternal line to the midclavicular line in the fourth interspace.

The post-mortem examination followed immediately the palpatory percussion, the findings of which were marked with the blue pencil on the skin of the cadaver. On opening the chest wall, no free fluid is found in the pleural cavities, the lungs prevented from collapse are firmly adherent to the anterior chest wall as follows: The sternum being replaced, the position of the heart, with regard to the *outlines* of the *heart*, are ascertained, which are *perfectly identical*, namely, extending from the midsternal line to the fourth interspace intersected by the midclavicular line. As in the former case, the palpatory percussion findings tally well with the post-mortem findings on the right side. The *upper lobe* of the *right lung* is bound down by dense *adhesions*, which pass along the *second interspace* anteriorly. The surface of the lung is irregular and studded by numerous dense nodules, of varying size, some of them from  $\frac{1}{4}$  to  $\frac{3}{8}$  of an inch in diameter. In the *third interspace* and *outside* the *midclavicular line*, extending toward the *anterior* and *midaxillary line* a *nodular infiltration*, but *no adhesions*, are

noticed, coinciding with the dulness mapped out previous to the opening of the thoracic cavity. Corresponding to the dulness extending from the right sternal border across the *fourth interspace*, a few adhesions are noted, but as cause of the change of sound numerous *tubercular nodules* are found just in that area. This nodular infiltration extends from the right *axilla* to the *right lower border* of the lung, following closely the direction of the *tenth interspace* and the *upper border* of the *tenth rib*. Right above this area limited by the *angle* of the scapula above and from it, the lung tissue is *soft*, crepitant and without nodular appearance. From the *7th interspace upward*, involving the *upper half* of the *right lower lobe*, as well as the *middle lobe* and particularly the *upper right*, lobes are firm, covered by a *thick pleura*, dense adhesions between the incisuræ. In the lower half of this agglutinated mass, including the *apex* of the *right lower lobe* and the *lower border* of the *upper lobe*, a *dense tubercular infiltration*—as in *caseous pneumonia*—is encountered. The upper half of the lung is *honeycombed* by numerous multilocular and intercommunicating cavities, partly filled with a creamy, hemorrhagic detritus. As regards the findings in the left lung anteriorly as substratum of the dulness encountered in the first interspace, we had again a *fan* (Dunham's) of dense adhesions which followed the direction of the *second rib*. Above and just between the left midclavicular line and the left sternum the ordinary lung tissue is substituted by a *nodular infiltration* of *conglomerating* tubercles. In the first interspace, *outside* from the *midclavicular line*, a white *scar* is found, and on section a *large cavity* is found, the walls of which are smooth and constituted by a *dense layer* of *connective tissue*. Below this area, the *inner border* being constituted by the *midclavicular line*, numerous large *tubercles* and *nodules* are found which are situated in the *outer half* of the *second, third and fourth interspace*. The *inner edge* of this infiltrated mass is formed by the heavily involved *lingual tip* of the left lower lobe anteriorly, revealing on section an *aspiration tuberculosis* with *caseating centers*. This tubercular infiltration extends toward the *axilla* and reaching in the posterior axillary line as high as the *lower border* of the *eighth rib*, then following the upper border of the 9th rib right to the spinal column, a small strip of *non-infiltrated* and soft pulmonary tissue is found which is situated

*just below the angle of the scapula.* The *lower third* of the *interscapular* region is again occupied by a *densely infiltrated area* of tubercular pneumonia. In the 5th and sixth interspace in the left interscapular region no further tubercular infiltration is encountered, the lung tissue being soft and elastic. Corresponding to the *lower border* of the *fourth rib*, and over the remainder of the left upper lobe, *dense adhesions* and *numerous tubercular nodules*, scattered all over, with only a very small amount of emphysematous pulmonary parenchyma, is found, which changes must account for the impaired percussion sound. In reviewing the case, a comparison of the physical findings with the post-mortem pathological changes again show a close similarity. Although the pathological substratum of an impaired percussion area may be a tubercular infiltration, nodular infiltrations alone or complicated with pleural thickening and adhesions we cannot expect from one step in the technic of physical diagnosis to reveal all the pathological changes. As is shown by this case, we can determine by palpatory percussion the extent of the lesion very well, but we cannot expect to dissociate the dullness projected from the diseased organ upon the body surface into its histological or pathological constituents. In the foregoing cases I had no opportunity of making a physical examination, whilst the patients were still alive, without any further information from the members of the staff about their physical examination the corpse was examined by myself, just with the purpose to test the reliability of the palpatory percussion method. Although it might be of interest to prolong the discussion of similar cases, it will be only one more case in which I examined the patient three weeks before exitus and only again on the post-mortem table. This case is of more interest, as stereoscopic X-ray plates from the cadaver lungs could be taken for closer study of the lesions.

Case III. Mr. W. H., age 49, a white laborer who had been suffering from pulmonary tuberculosis for the past 9 months. A brief outline of the physical findings may precede the post-mortem percussion. On the *right side anteriorly* impaired percussion extends from the right apex to the *second interspace* as far as the inner half of the space bordered by the midclavicular and anterior

axillary line, then sloping downward toward the right axilla over the third and fourth interspace. On auscultation, *low-pitched, cavernous* breathing and crackling rales, pectoriloquy No. 3, are located over the dull areas in the first and second interspaces. In the *third* interspace anteriorly harsh *breathing* bronchophony, are encountered, in the fourth interspace cogwheel breathing, friction rubs and bronchophony are noticed. *Posteriorly* the right lung gives impaired percussion sound, including supra and infra-scapular fossæ, and interscapular region as far as the sixth interspace. Over this area on auscultation, from the *third interspace* upward, *cavernous, low-pitched* tubular breathing and pectoriloquy No. 3 are encountered, whilst below this area as far as the 6th interspace expiration is prolonged, and over the middle of the scapula fine crepitant rales are added to a distinct pectoriloquy. The seventh interspace of the right interscapular region is normally resonant and reveals normal breath sounds, but immediately below a *triangle of impaired percussion* sound exists, over which area the *expiration* becomes harsh, prolonged, high-pitched and with distinct pectoriloquy No. 2. This triangle situated over the 8th and partly over the 9th interspace is separated by a narrow zone of normal resounding tissue from an *absolute dulness*, which in the *tenth* and *eleventh* intercostal space ascends *toward the right axilla*. On auscultation the breath sounds are very *distant*, and *faint*, no whispered voice being perceptible over the same area. Over the *left lung anteriorly* a dulness extends from the left apex involving the *first* and *second* intercostal spaces, which *outside* from the left *midclavicular line* slopes over the *third* and *fourth* interspace to the left axilla. Over the outer half of the first interspace a small area of distinct *hyperresonance* is found in midst of the neighboring dulness. Harsh *tubular breathing* inspiratory and expiratory, numerous crackling rales and *pectoriloquy* No. 3 are encountered over the area of dulness, the *expiration* toward the axilla, though *prolonged*, is *less* marked and so is the pectoriloquy. Arising from the left pleural sinus a *dulness* covers the *fourth interspace*, extending into the left axilla with a roughened expiration, prolonged expiration in the third interspace, which becomes very *indistinct* in the *fourth* interspace. *Posteriorly*, the *left apex* is *dull*, which percussion note changes into normal resonance in the *fifth* interspace in

the interscapular region. Corresponding to these findings, harsh *prolonged expiration* changes upward from the fourth interspace into *tubular breathing*, over the upper part of the left apex, with corresponding *transition* from pectoriloquy No. 1 to pectoriloquy No. 3. Harsh *expiration* over the *middle third* of the scapula, pectoriloquy No. 1, fine crepitant inspiratory and expiratory rales are noted. A *triangular-shaped dulness* is mapped out, which extends from the *upper border* of the *7th rib downward* to the *7th, 8th and 9th interspace*, the outer and upper part of which are covered by the *tip* of the lower angle of the left scapula, over which area *harsh breathing*, pectoriloquy No. 2, exists. The *9th interspace* is found *more resonant*, the *10th*, and including the left basis beyond, appear *impaired*, the breath sounds becoming only *faint* and *indistinct*. With such an extensive pulmonary involvement, the fatal prognosis was soon to become true, the patient died 3 weeks later after a very septic course of temperature and the post-mortem examination was soon made. In the findings of the palpatory percussion, which were mapped out immediately preceding the opening of the chest over the *left lung anteriorly*, a *dull note* with *increased resistance* to touch is found over the apex, the *first, second interspace*; the *third interspace* just outside of the left midclavicular line, extending over the *fourth intercostal space* toward the *left basis* in the axilla, leaving only a very small section of normal percussion area in the third interspace, to the inside of the midclavicular line and the left sternal border. Posteriorly the *entire left basis* is *dull*. A little *above the lower fourth* of the left scapula the interscapular space, corresponding to the direction of the *6th interspace*, a marked increase of the *resistancy* is encountered which is found to extend to the *seventh and eighth intercostal space*, merging into the dull percussion area of the *left basis*. *Above* this zone, following the *sixth interspace* in the left interscapular region, *normal percussion* note and resiliency of the tissues are encountered. The *entire area above* from the *fifth interspace*, including the left apex, is *dull* and *very resistant*. The *right lung* on palpatory percussion gives the following changes: *Dulness* all over the *first and second interspace* anteriorly, also *dulness* over the *third, fourth interspace* extending from the *right midclavicular line* toward the *right axilla*, leaving only a small area of normal



percussion note in the space limited by the right midclavicular line and the right sternal border in the *third* and *fourth* interspace. *Posteriorly*, a dulness and marked increased resistance are encountered which extend *upward* from the *seventh* interspace all over the *right* apex. The maximum of resistance is found over the *seventh* and sixth interspace. The cardiac dulness extends from the left midclavicular line and the fifth interspace to the right border of the sternum.

In opening the chest wall, the lungs do not retract, particularly the *right lung* is firmly *adherent* to the *costal pleura* extending anteriorly to the *second intercostal space*. The lung area corresponding to the *third* and *fourth interspace*, *outside* of the *midclavicular line* and extending *toward* the *axilla*, shows a large number of *tubercular nodules*, whilst only a *small area* of *normal* and *crepitant pulmonary* tissue is found *beneath* the *fifth rib*. *Posteriorly* the *lower third* of the *right lower lobe* is found normal, but the *apex upward* from the *upper half* of this lobe is densely *infiltrated* by a *pneumonic mass* of caseating tissue. The *middle right* and the *upper right lobe* are *densely studded* by large-sized tubercles. On section the *upper lobe* is *honeycombed* by a system of several cavities, irregular in shape, containing a granular serous fluid, besides many fair-sized gray caseous tubercles. In the *left pleural cavity* about 500 Cc. of a *serous fluid* was found, only a *few recent adhesions* being present. All over the *left lobe*, *anteriorly*, hard *infiltrated* nodules were palpated, which coincided *exactly* with the extent of dulness found in the *first* and *second interspace* anteriorly. The *dulness* encountered in the *third* and *fourth interspace*, limited anteriorly by the *left midclavicular line*, was found due to a *heavy pneumonic infiltration* which involved the *lingual tip* anteriorly of the left lower lobe. *Posteriorly* the *upper left lobe* was found transformed into a *system* of intercommunicating *cavities*, interspersed by nodular tubercles. Only one small section of the lower *third* of the left upper lobe was found normal as to consistency and corresponded to the *normal resonant sound* encountered in the sixth interspace. Of the *lower lobe posteriorly* about *one-half* was found *solidified* by a *bronchopneumonic process* involving the *apex* of that lobe. About the heart size compared with the size ascertained on percussion a *close concordance* was found,

with a hypertrophied and dilated right ventricle, a small and flabby left ventricle, showing myocardial degeneration.

In the discussion of these percutory and post-mortem findings, I will not enter upon the auscultatory findings in vivo compared with the pathological changes revealed, suggestive signs of cavitation were present, particularly over the right apex, to a lesser extent over the left apex, but to conclude from the auscultatory signs upon the size and exact location of the cavity means a delicate procedure, which on the post-mortem table may not materialize the anticipations of the expert clinician. The palpatory percussion used in this instance revealed a close analogy with the post-mortem findings as to the extent of the pathological changes. Of course, whenever we encounter pathological changes in the quality and tonality of the percussion note, a change in the resiliency of the tissues intervening between the palpating finger and the diseased area of the lung, it is difficult to decide whether the change is caused by a thickened pleura, by a nodular, tuberculous or bronchopneumonic consolidation from the palpatory and percutory impression alone. Auscultation, whispered voice, may help in such a decision just as in a jury case we try to ascertain all the facts before reaching a plausible explanation. In presenting these examples as a support to the value of palpatory percussion in physical diagnosis in the past decade of years of practical experience, it is my impression that, in teaching students and postgraduates, the method should be taught as promising for a better insight into the frequently obscure chest conditions. Personally, when I first got acquainted with the method, considerable time and practice was necessary for the proper utilization of the touch method. The question is not so much to cause by a strong percussion stroke a vibration of a large area including in it diseased and normal tissue, but just to compare the sound between diseased and normal parts. Provided that the present communication and study will stimulate further comparison of well-accredited methods of physical diagnosis with newer means of approaching the truth for the benefit of our patients, this will add to our professional efficiency.

#### SUMMARY

1. For the diagnosis of chest diseases we must resort to all means

available to secure the diagnosis, and as often as permissible should urge a post-mortem examination in fatal cases.

2. Besides the ordinary strong percussion, palpatory percussion should be taught to the student and to the postgraduates.

3. In pulmonary diseases we must not be satisfied with the statement that the patient has got such and such a disease; in pulmonary tuberculosis we must try to ascertain by all means the extent of the lesion.

4. With the proper technic of palpatory percussion, it is possible to approach very closely the post-mortem changes so far as the involvement is concerned. As to the cause of the change in the resistance and in the character of the percussion note we are frequently unable to determine in vivo by percussion alone whether the dulness is caused by pleuritic adhesions, nodular tubercles, or bronchopneumonic caseous tuberculosis, or by edema.

5. The outspread of the tuberculous process into the apices of the lower lobes is recognizable or suggested by a *distinct triangular area of dulness in the interscapular space*—similar in shape to the Grocco's sign in pleurisy, with possible auscultatory signs present.

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### PROGNOSIS IN PULMONARY TUBERCULOSIS

By EDWARD O. OTIS,

Professor of Pulmonary Diseases and Climatology, Tufts College Medical  
School  
Boston

Louis, the illustrious disciple of the still more illustrious Laennec, declared nearly one hundred years ago in his "Researches on Phthisis" that phthisis (tuberculosis) "almost invariably terminated fatally," whereas at the present day we say with more or less truth that it is one of the frequently, or the most, curable of chronic diseases.

What has happened in the meantime to produce such a radical change in the attitude of the physician toward this disease? There are three main causes: First, the discovery of the specific organism of the disease, the tubercle bacillus; second, early diagnosis; and, third, timely treatment by the hygienic-dietetic, or "open-air" method.

In spite of this more hopeful aspect of the outcome of pulmonary tuberculosis, the mortality from it, as we all know, is still very great, only equalled by that other pulmonary affection, pneumonia and cardiac disease. The disease is still, as Louis so many years ago said, "one of the most relentless enemies of the human race."

On the other hand, however, experience has shown that it is arrested not only in its incipient stage but in more advanced stages, and sometimes even without any methodical treatment.

How are we to be guided in forecasting the result in any individual case? What indications are there which in one case lead us to give a doubtful or unfavorable prognosis and in another case to give a favorable one?

In the first place, one can postulate in general that the earlier the case comes under treatment the more favorable the prognosis. One, however, sees many exceptions to this general proposition. Treatment may be unable to develop any substantial or lasting resistance, and in spite of the most skilful application of the open-air treatment, the case, even from a slight beginning, may go on uninterruptedly from bad to worse, to a fatal termination. As someone has said, the disease, so to speak, may be advanced from the beginning. The only hope of a favorable outcome must depend upon the development and maintenance of a good and capable resistance, and hence the only reliable prognosis must be founded upon the observed results of treatment.

There are, however, many valuable indications to guide one in making a provisional prognosis from the first time the case is seen. Of general indications an important one is the previous life and habits of the patient. If he has lived a regular, hygienic life under favorable conditions of fresh air, rest and good food, and the avoidance of all excesses and yet develops active tuberculosis, it is a fair inference that his normal resistance is low, and, hence, the

prognosis is less favorable than with one who has lived and worked under unwholesome conditions either in or out-doors and has been deprived of those favorable influences under which the first patient lived. The change to the wholesome life is vastly greater and may be assumed to produce more favorable results than in the former case.

Again, the character and intelligence of the patient play an important rôle in the prognosis. A tractable, intelligent, persistent patient who will co-operate with his physician obviously offers a better prognosis than a self-willed, ignorant one. "One cannot cure a fool," as someone has said.

Other evident general conditions which influence the prognosis are the habits of the patient, particularly as to the use or abuse of alcohol, his social and domestic relations, occupation and family predisposition (although there are varying opinions as to the importance we should attribute to the family history), and the pecuniary ability to procure treatment.

Coming now to special indications as influencing the prognosis we have: (a) The character of the onset of the disease. A very acute beginning, as indicated by marked symptoms, such as fever, night sweats, rapid loss of weight and strength, anorexia, is an unfavorable omen, while an insidious onset is of no especial prognostic significance. (b) Again, the rate of progress in relation to the duration of the symptoms; if there is evidence of rapid extension of the disease, or both lungs are involved at an early period the prognosis is unfavorable. (c) The character of the symptoms; if these are marked and progressive, the prognosis is unfavorable, while if they are slight and retrogressive, it is favorable. (d) Increase of moisture in the diseased portion, as indicated by the increase and size of moist râles is an unfavorable indication even if there is general improvement. (e) Any serious complication, either tuberculous or non-tuberculosis, such as tuberculosis laryngitis, diabetes, or other chronic or acute diseases, injures the prognosis. Pleurisy with effusion, however, in the course of the disease, I do not consider to alter materially an otherwise favorable prognosis. On the contrary, it appears not infrequently to exercise a favorable influence upon the subsequent course of the disease. (f) Pregnancy in an active stage of the disease renders the prognosis far

more unfavorable, as after the confinement the disease is likely to and generally does become more active, although there are occasional exceptions. (g) Various acute diseases, such as influenza, bronchitis, typhoid fever, pneumonia, and whooping cough or measles in children, occurring shortly before the advent of the tuberculosis, render the prognosis rather more unfavorable, as the resistance is depressed by the previous infections. (h) If the tuberculous individual is unable to eat and digest the requisite amount of food and be nourished by it, it is a very unfavorable factor in the prognosis, for, as someone has said, "A consumptive who cannot eat is doomed."

The pulse rate, the temperature and the weight are cardinal points in estimating the prognosis; a low or moderately low pulse rate is always a favorable factor and when associated with a normal temperature and increase or at least no loss of weight from the normal, enables one to give a very favorable prognosis.

As to the influence of hemoptysis upon the prognosis, occurring either as an initial symptom or later in the course of the disease, it is not to be considered necessarily an unfavorable prognostic symptom, particularly as it occurs in 50 per cent. or more of all cases, many of whom make a perfectly uneventful recovery. If, however, the hemorrhage is accompanied with fever which persists and other symptoms of increased activity with the extension of the local area of disease, it is, of course, an indication that the disease is progressive and therefore an unfavorable prognostic factor. The psychic effect of a hemorrhage, however, is sometimes so marked that the patient loses his morale and equanimity of mind and, in consequence, the "will to get well" is seriously weakened and the courage for the conflict dampened.

Finally: The prognosis in any individual case, in the last analysis, will depend upon the resistance of the patient to the toxic effect of the tubercle bacillus. As with other infections it is the toxemia which kills, not the destruction of lung tissue. If the resistance can be effectively developed and maintained, the result will be favorable. Sometimes this occurs speedily and decisively, but more frequently it takes place more slowly and with many recessions; and, unfortunately, it never occurs, try as we will.

To summarize: The symptoms which render the prognosis at

the time unfavorable are (a) a rapid and slow tension pulse; (b) continued fever; (c) steady and rapid loss of weight; (d) anorexia and malassimilation; (e) loss of strength; (f) marked dyspnea; (g) evident and rapid extension of the local destructive process.

On the other hand, the symptoms which render the prognosis favorable are (a) no fever; (b) a quiet pulse and nervous system; (c) weight not diminishing or increasing; (d) a good appetite and digestion; (e) strength not materially lessened; (f) the local process limited or not advancing.

The physical signs alone are a less trustworthy prognostic guide than the symptoms alone. It is not the lung involvement which leads to the fatal issue, but the active and continued toxemia of the tuberculous infection. Improvement and apparent arrest are not infrequently observed without much if any change in the physical signs, although this is not the usual course. When we see, therefore, a patient entirely devoid of any symptoms and, so far as his sensations go, in good health and strength, we can say that he is at least, economically well even though we detect rales in the lungs from examination to examination. A word with regard to the prognosis of an arrested case—what is the prospect of maintaining the “arrest” or “cure” the remainder of one’s life? Some cases never relapse. The patient resumes his real mode of life and occupation and forgets that he ever had the disease.

Other cases, especially if one reverts to a former unwholesome life, relapse, and the last condition is worse than the first.

With the majority of cases eternal vigilance is the price of freedom from a recurrence. It is only by a strict adherence to the wholesome plan of life, learned while under treatment, the avoidance of all excesses, particularly overwork, worry, the neglect of proper nourishment and rest, that one can hope to maintain the “arrest” obtained through much labor and self-denial. He must ever remember that he is probably more susceptible to another attack of the active disease than if he had never suffered from it. He must live a watchful God-fearing life and be profoundly thankful that in so doing he had a good prospect of remaining well.

## UPON THE USE OF TUBERCULIN IN THE DIAGNOSIS OF EYE LESIONS

By C. A. CLAPP

Baltimore, Md.

Since the more recent works have shown that many eye lesions which were previously diagnosed as idopathic, are in reality tuberculous, and that while clinical history and macroscopic appearance may point to the lesion being tuberculous, every careful diagnostician has been chagrined at the *clinically* tuberculous lesions showing a strongly positive Wasserman Reaction and being entirely cured under antisppecific treatment, whilst conversely a lesion which appears clinically to be specific is uninfluenced by antiluetic treatment and then later show a focal reaction following a tuberculin test.

The methods in use at the present time in arriving at a conclusion that the lesion is tuberculous are:

- 1.—The ophthalmic reaction (Wolf-Eisner, Calmette).
- 2.—The cutaneous reaction (Von Pirquet).
- 3.—The sub-cutaneous diagnostic injection of old tuberculin (Koch).
- 4.—Focal reaction following increasing therapeutic doses.

Taking these methods in detail as to their value and adaptability we desire to make the following comments:

(1) The ophthalmic reaction with tuberculin, may show in general work that the lesion is in an active stage, but should be used with great caution, and certainly never in an eye which has a suspected local tuberculous lesion. Violent reactions have followed its use with permanent loss of vision due to corneal ulceration.

(2) The cutaneous reaction of Von Pirquet has no contra indications, as damage to the eye never results, but on the other hand very little knowledge of positive nature is obtained. If the reaction does show positive, it may be taken as suggestive, but since about seventy-five percentage of adults show some reaction, even its suggestiveness diminishes with increased age of the patient. If the reaction shows negative, it is also of little value as experience has shown that cases which are negative to the skin reaction, very frequently show focal reaction, under tuberculin therapy; therefore,



Von Pirquet's reaction either positive or negative is of very limited value in the differentiation of the etiology of eye lesions.

(3) The sub-cutaneous injection of T. O.

For convenience of discussion this reaction may be subdivided into (a) General reaction; (b) Focal reaction.

(a) *General reaction:*

If following a diagnostic dose there is a febrile reaction of one or more degrees Fahrenheit coming on within six or eight hours and especially if accompanied by a local reaction it is very good evidence that there is a tuberculous lesion some where, but if no focal reaction is produced, the tuberculous lesion is probably outside of the eye. In other words, I can hardly conceive of an eye lesion which would liberate sufficient toxins into the general circulation as to cause a febrile reaction, without causing some focal reaction. The dangers of a large diagnostic dose upon eye lesions, I propose to show more in detail later, but I wish to state here that the danger to sight is very great.

(b) *Focal reaction:*

This is the reaction of real value to the ophthalmologist. Hamman and Wolman say in their "Tuberculin in Diagnosis and Treatment": "This is the reaction of most value, but seldom do we have an external lesion where the reaction can be seen." Thus is tuberculin of so much more value to the oculist, as he has direct observation of the lesion, either in an external trouble, or if internal in most cases under direct view by means of ophthalmoscopy.

Now as to what constitutes a reaction. If the lesion is corneal there is seen an increase in the circumcorneal injection with an extension of infiltration into the cornea. It is not necessary to have a reaction that a layman can see across the room to be called a focal reaction, but as one watches the eye, slight increase in the circum-corneal redness after each injection can be seen, or in oedema of cornea or extension of infiltration into deeper layers. If iritic, there is an increase circum-corneal injection with more numerous deposits on Descemet's membrane and some increase in cloudiness of aqueous; if choroidal or retinal the lesion shows greater activity, in increased oedema and cloudiness of the vitreous, and possibly fresh hemorrhages if the blood vessels be chiefly affected. All these

macroscopic changes may be from very slight to very pronounced in degree.

**Size of dose.** Since publishing the article "A Report of Six Cases treated with Tuberculin, including cases of Keratitis choroïditi and cycliti," in "Annals of Ophthalmology, July, 1916," I have had repeated inquiry as to the average size of the diagnostic dose to be given. I desire to lay particular stress upon the fact that there is no such thing as an *average* size diagnostic dose. If the lesion under investigation is corneal, where the vascular supply is very deficient, then a large diagnostic dose, one to ten mg., can be used with impunity, as these lesions show a very great indifference to reaction. While one mg. is usually sufficient to provoke a reaction and seldom is it necessary to go beyond this amount, yet occasionally one may find a focal reaction with the larger dose of ten mg. which has not reacted to the smaller doses. When the lesion involves the iris, a smaller initial dose should be used, depending upon the activity of the lesion. In an acute condition a one-half mg. T. O. will not produce too violent a reaction, while in the more or less chronic conditions, a painful or harmful reaction will not result from even five mg. But when we are dealing with a choroidal or retinal lesion and especially if near the macula, then as a rule only the smallest doses should be used, and should the lesion be very acute, I deem it most dangerous to use any diagnostic dose, and would strongly recommend that the eye be put at rest; general hygienic measures instituted and *wait* until the lesion becomes sub-acute or chronic before using a diagnostic dose, and even then it is better to start with exceedingly small therapeutic doses and increase until a general or focal reaction results. In other words, we should never allow our scientific zeal to render an absolutely correct diagnosis, overcome our judgment as to conservative treatment.

(4) The focal reaction following therapeutic doses:

Under this heading come a fairly large group of cases that one sees in our dispensaries and occasionally in private practice and those are the so-called neglected cases, which have allowed damage to sight to occur before calling for help, or that class of patients who make too frequent changes in their medical attendants. When such a case comes, with the cornea, aqueous, pupil or vitreous, so

cloudy that the exact location, extent, or acuteness of the lesion, cannot be determined; or in those acute cases, where the lesion is centrally located a diagnostic dose would be decidedly dangerous. If after careful investigation all other possible sources of etiological factors prove negative and all points of focal infections be eliminated, it is then highly advisable to start in with very, very minute doses of the tuberculin sub-cutaneously gradually increasing the dose until some focal reaction is noted, or until there is seen some actual improvement in the condition. Here again one could with justice ask what we mean by a very minute dose. If T. O. is being used 1 : 500,000 mg. is not too small as the initial dose, while with the T. R., which is the preparation we have had most experience, may be used in a little larger dose; 1 : 300,000 mg. never having proved harmful in our work. One frequently sees a beginning improvement in the eye before a focal reaction, but in these cases almost without exception the focal reaction appears sooner or later.

After one has watched several of these lesions that have persisted for months with no improvement under various lines of treatment suddenly begin to improve after tuberculin injections, it is impossible to be convinced that the changes are entirely coincidental.

These observations have therefore brought us to the following conclusions:

#### CONCLUSIONS.

(1) That a differential diagnosis between the different eye lesions as to etiology cannot be made from clinical observations alone.

(2) That if all other tests are negative and we obtain a focal reaction we are reasonably certain in our diagnosis.

(3) That the size of the diagnostic dose is not the same but varies widely as to the location of the lesion, and as to its stage of activity.

(4) That occasionally the media are so cloudy, or the lesion so acute, that no diagnostic dose should be given, and in such cases a minute therapeutic dose should be given and gradually increased until a reaction is produced, or until we eliminate the tuberculous element as an etiological factor.

(5) Never be so aggressive in the diagnosis of etiological factor as to permanently damage the sight.

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"Dr. Bing gives us, in the present book, a good manual for the topographical anatomy of the brain, which will assist the physician in localizing clinically diseases and injuries of the central nervous system. The translation seems to be very well executed. The book is one of the few which are of practical value."—*New York Medical Journal*.

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